REPORT

OF THE

NEW YORK STATE VETERINARY COLLEGE

AT CORNELL UNIVERSITY

For the Year 1916-1917

TRANSMITTED TO THE LEGISLATURE JANUARY 31, 1918

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JANUARY 31, 1918

REPORT OF THE NEW YORK STATE VETERINARY COLLEGE

STATE OF NEW YORK — EXECUTIVE CHAMBER

ALBANY, *January* 31, 1918

TO THE LEGISLATURE:

I have the honor to transmit herewith the Annual Report of the New York State Veterinary College at Cornell University for the fiscal year ending June 30, 1917.

CHARLES S. WHITMAN.

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REPORT

January 15, 1918

The Honorable Charles S. Whitman, *Governor of the State of New York, Albany, N. Y.:*

Sir.— In accordance with the requirements of chapter 689 of the Laws of 1897 providing for the administration of the New York State Veterinary College, I have the honor to submit on behalf of Cornell University the accompanying report of the operations of the college for the year 1916-17.

The great world-war in which the United States is now a participant gives to the subject of the flocks and herds an importance it has never before possessed in the history of the country. The western European Allies (Great Britain, France, Belgium, and Italy), were, even in times of peace, large importers of cereals and meats, and the war having depleted their own supplies America must make good the shortage. Their herds have been seriously cut into since the beginning of the war by the lessened production and import of feed grains and fodder for their support and by the necessity of eating into the capital stock to meet the pressing demands for an increased ration of meat and animal fat of millions of men who have been transferred to the armies, to the war factories, and to other positions demanding severe physical exertion. This reduction of their herds by these causes means, of course, a lessened reproduction of animals. And while a cereal shortage may be made good in one year it takes two or three years for the natural replacement of depleted herds. A few figures will make the situation clearer. The cattle, sheep, and hogs of the western European Allies of 1913 were over one hundred million head. At the beginning of the year 1917 they were estimated at about seventy-five million; and by the beginning of the year 1918, if the decline in France followed for the last twelve months the rate of decline which it followed for the three years preceding, that country cannot possess more than twenty-six million head as compared with thirty-eight million before the war.

The effects which the war has produced in diminishing the farm animals in European countries cannot fail, though in a lesser degree, to be operative in the United States. The high price of feed grains and fodder discourages the increase by the farmer of his flocks and herds, and the increased demand made for meat and animal fats will tend to a diminution of the existing numbers. It is of the first importance, therefore, that every effort should be made in this and other states to maintain the flocks and herds in undiminished numbers.

According to the Year Book, Department of Agriculture, for 1916, the number of animals in this State and their value are, with the exception of poultry, given as follows:

	Number	Value
Milch cows	1,539,000	$101,574,000
Other cattle	939,000	29,109,000
Horses	609,000	84,651,000
Mules	4,000	592,000
Sheep	840,000	7,056,000
Swine	759,000	11,157,000
Total		$234,139,000

	Number
Poultry (Farm Census, 1915)	17,976,935

Apart from the effects of the war the greatest losses to the live stock industry are due to the diseases of animals. The direct property losses from the diseases of animals in the United States in 1915 were estimated by the Secretary of Agriculture to be $212,000,000. The indirect losses cannot be estimated at all, but they are obviously very considerable. The losses from each of the different diseases as given by the Secretary are shown in the appended table, which also shows the estimated proportionate losses to the State of New York:

Disease	Loss in U. S.	Percentage of Animals in N. Y.	Estimated loss to N. Y.
Hog cholera	$75,000,000	1 per cent swine	$750,000
Texas fever	40,000,000	8 per cent cattle	None
Tuberculosis	25,000,000	6 per cent dairy cattle 2 per cent other cattle.	2,000,000
Contagious abortion	20,000,000	6 per cent dairy cattle	1,200,000
Blackleg	6,000,000	Cattle	Slight
Anthrax	1,500,000	13 per cent all animals	195,000
Scabies	4,600,000		Slight
Glanders	5,000,000	2 per cent horses	100,000
Other diseases	22,000,000	13 per cent of all animals	2,860,000
Parasites	5,000,000	13 per cent of all animals	Small
Poultry diseases	8,750,000	7 per cent of all poultry	612,000
Estimated total loss for New York State			$7,717,000

These losses to New York State would no doubt be much heavier than they are but for the successful work accomplished by the veterinarians of the State. And if the losses are to be reduced now and in the future it can only be through the better training of veterinarians to serve the live stock districts of the State. These veterinarians must be qualified not only to treat successfully the simple diseases but also to recognize promptly the infectious diseases and to assist live stock owners to protect their animals against them.

These facts show the importance to the State of thorough veterinary education. A score of years ago a man was graduated as a veterinarian with almost no general education, and with two years or even one year of professional training. In the State Veterinary College at Cornell University the course has always been one of three years, and in 1916 it was lengthened to four years; and the preliminary education required for admission is at least that of graduation at a high school.

It is obvious also that State support is absolutely essential for the provision of such a thorough education. The enormous increase of knowledge in recent years concerning the cause, treatment, and control of animal diseases has made the veterinary course so extensive and so costly to provide that it cannot possibly be given by any institution supported solely by the fees of students. From its inception veterinary education was considered in most European countries to be a function of the State. And in 1894

New York State recognized and adopted this principle through the foundation of the State Veterinary College at Cornell University.

While the conditions call for better training of veterinarians than ever before and for a higher grade of scientific work, the number of veterinarians needed in the State of New York is less than formerly. This is due to the displacement, especially in the cities, of the horse by the motor car. In 1909 there were 1,121 registered veterinarians in New York State; in 1916 the number had fallen to 848 and in 1917 to 763. The decrease is especially marked in the large cities. In New York city (including the Bronx) there were in 1909 181 licensed veterinarians and in 1917 only 104. In Brooklyn there were in 1909 96 and in 1917 only 62. In Buffalo there were in 1909 31 and in 1917 only 17. There is no complaint of inadequacy of numbers at the present time. On the contrary it is the general opinion of agriculturists, breeders, practitioners, and other experts that there are veterinarians enough for the present needs of the animal industry of the State. Approximately 800 qualified veterinarians are able to care for the present animal population of the State as set forth above. And from twenty-five to thirty new graduates annually will fill the vacancies caused by the retirement from natural causes from the ranks of these 800 veterinarians.

As Dean Moore has pointed out in his report, the shifting of the center of veterinary activities from the city to the country calls for a much more comprehensive course of instruction than has been given heretofore. A generation ago the veterinarian was known as a "horse doctor." But to-day, while the number of horses in the city has greatly declined because of the general use of the automobile, the diseases of cattle, sheep, swine, and poultry are constantly making heavier demands upon the veterinarian, and from an economic point of view they are of the greatest importance, especially in time of war. The graduates of the State Veterinary College, four-fifths of whom come from homes in small cities and rural districts of the State, in general remain within the State of New York. Of the 390 living graduates of the college, 246 are in this State. Of the 32 students who received their degrees in June, 1917, the majority also intended to practice in New York State. The declaration of war, however, changed their

purpose, and 15 of them took and passed the examination for officers in the Veterinary Corps of the Regular Army, and practically all of the rest took the examination for the Veterinary Officers Reserve.

Besides the work in instructing students the faculty of the college have always devoted much time to research, which was defined as one of the objects of the college in the law providing for its administration. At the time the college was opened research work on animal diseases had just begun in this country, and biological products were not used for diagnostic or prophylactic purposes and instruction in veterinary medicine had not been placed on a university basis. The annual reports of the Veterinary College show that the members of the faculty have made important investigations and discoveries in the field of animal diseases, including diseases of hogs, cattle, horses, sheep, poultry, the results of which have been of enormous financial value to the State. The college has always co-operated with the Commissioner of Agriculture in undertaking the study and, if possible, the solution, of scientific problems which arose in connection with his work. In the field of diagnosis alone thousands of specimens have been examined in the pathological laboratory of the college for the veterinarians in the employ of the Commissioner of Agriculture and for practitioners. And the college has also prepared and supplied on request to veterinarians and animal owners great quantities of mallein, tuberculin, anthrax vaccine, and anti-hog-cholera serum and other diagnostic and prophylactic biologic agents.

The work of the college is carried on in buildings and hospitals which have been provided by the State at an aggregate cost including equipment of less than $350,000. Supplementing the buildings and equipment provided by the State, the trustees of Cornell University have set aside a farm of 100 acres for the use of the Veterinary College for conducting experiments in connection with the study of animal diseases and to provide a suitable place for the production of anti-hog-cholera serum. The use of this land has made it possible to carry out many investigations that otherwise could not have been made.

A new wing and laboratory building are now urgently needed to carry out the original architectural plan and to provide accommodation for the constantly increasing research work which the college undertakes.

In November, 1917, Dean Moore presented by request to the Board of Trustees of Cornell University a report on "How the New York State Veterinary College at Cornell University is Meeting the Veterinary Needs of the State and Country." That report, which was a very instructive one, concludes with the following summary:

"The efforts of the State to meet the actual veterinary needs of its animal owners may be summarized as follows:

1. The State has established a Veterinary College under the supervision of Cornell University in which it has already invested $346,500 in buildings and equipment.

2. The State is now able to furnish at the Veterinary College at Cornell University veterinary instruction to enough students to insure a graduating class of from 35 to 50 men each year. This college has graduated during the last five years an average of 32 students annually.

3. For the protection of its live stock interests the State requires about 25 new veterinarians each year. Its quota for the federal government requires from 5 to 10 more.

4. The State Veterinary College has made many important investigations on animal diseases and is now conducting others that promise to be of much value to the veterinarians and animal owners.

5. This college is able to furnish all of the diagnostic and prophylactic biologic products that the State requires. It also is doing all of the laboratory diagnosis work called for by the Commissioner of Agriculture and the practising veterinarians of the State.

It is hoped that in the near future this institution may be completed and that funds will be forthcoming to insure steady progress in teaching and in the study of animal diseases. There is every reason to believe that the State, through the college, can satisfy all of its obligations respecting veterinary education and the preparation of practitioners."

I desire to endorse this statement and to commend it to the Governor and Legislature of the State as a basis of action in dealing with veterinary matters and in providing for the State Veterinary College at Cornell University in the winter of 1918.

I have the honor to be

Your obedient servant,

JACOB GOULD SCHURMAN,

President of Cornell University.

REPORT OF THE DIRECTOR

January 5, 1918

PRESIDENT JACOB GOULD SCHURMAN:

SIR.— I have the honor to submit herewith the report of the New York State Veterinary College at Cornell University for the fiscal year ending June 30, 1917.

The work has progressed satisfactorily and, on the whole, it has been more efficient than heretofore. The most noteworthy educational feature was the beginning of a four year curriculum. The necessity for lengthening the course of instruction had been recognized for a number of years but adequate clinical material for teaching practical medicine, including the diseases of breeding cattle, and operative surgery was not assured until now. The clinics having been organized and developed, it was possible at the beginning of the year to undertake more extended and, we believe, more efficient instruction for men who wish to practice veterinary medicine. The facilities for research in the basic subjects of anatomy, bacteriology, pathology and physiology are also improved.

The shifting of the center of veterinary activities from the city to the country calls for a much more comprehensive course of instruction than has been given heretofore. The diseases of cattle, sheep, swine and poultry have come to be of great importance economically. In order to prevent the losses which they occasion, thorough instruction must be given in the nature of these diseases and in the methods for their diagnosis and control. This calls for much expansion in the scope of the instruction that has been given heretofore in veterinary colleges. This institution is fortunate in being located on the campus of a great university from which it draws the most advanced knowledge in the basic sciences and further in being surrounded by a farming community which supplies a large clinic consisting of practically all species of domesticated animals which suffer from the diseases common in this latitude.

The advent of war caused much disturbance in the work of the

students during the last weeks of the year. It was thought best that they should continue their studies, and when graduated enter the army as veterinarians. However, a few went to officers' training camps and to the enlisted forces. Later, when the urgent appeal came for more labor to put in and harvest the crops, ninety of the students were given permits to do this work. The subject-matter which they missed will be made up during the present year. Special lectures and demonstrations are being given to aid the students in this additional task.

Faculty and Employees

It is essential to have for teachers men of experience as well as those of scientific and professional attainment. Unfortunately, during the last few years, the college has suffered serious loss through the resignation of members of the faculty who have felt obliged to accept places in other States because of the higher salaries.

In June, Dr. C. P. Fitch, Assistant Professor of Pathology and Bacteriology, resigned to accept a place in the University of Minnesota. This was a great loss not only to the college but also to the State. It requires years for men to develop to the point where they are efficient as teachers and research workers. This is especially true in the study of infectious diseases of animals where practical veterinary knowledge is a necessary adjunct. As a result of his resignation, the researches on infectious anaemia in horses, ovaritis in cattle and the parasites of sheep and other animals will have to wait for their completion until another man may be found who will be interested in these subjects. Meantime the losses from these affections among the live stock of the State will continue. Dr. H. E. Johnson, Instructor in Anatomy, resigned to go to the veterinary department of the Michigan State Agricultural College. Economically and educationally, it is unfortunate to lose teachers after they have reached the stage where they can do constructive work. Men filled with the spirit of scientific inquiry and devoted to teaching and research are not numerous in the veterinary profession and they are needed in New York quite as much as elsewhere in the country. Unless the State is willing to keep this class of workers, it must not hope to gain mastery over

the diseases that are costing millions of dollars annually nor expect to build up a veterinary college worthy of its animal industry.

The university furnished, in other colleges, instruction to the veterinary students in animal husbandry, chemistry, embryology, histology and physics. The veterinary faculty consisted of the following personnel:

Jacob Gould Schurman, president of the University.
James Law, emeritus professor of medicine.

Department of Anatomy

Dr. Grant S. Hopkins, professor of anatomy.
Dr. Earl Sunderville, assistant professor of anatomy.
Dr. Howard E. Johnson, instructor.
Dr. E. Stanley Stone, assistant.

Department of Materia Medica

Dr. Howard J. Milks, professor of therapeutics and small animal clinic.
Dr. W. E. Muldoon, instructor.
Mr. P. V. Dederick, student assistant.

Department of Medicine

Dr. Dennie H. Udall, professor of veterinary medicine and hygiene and superintendent of the ambulatory clinic.
Dr. F. F. Koenig, assistant professor of medicine.
Dr. Joseph B. Latshaw, instructor.

Department of Pathology and Bacteriology

Dr. V. A. Moore, professor of comparative pathology and bacteriology and dean of the college.
Dr. Samuel H. Burnett, professor of comparative pathology.
Dr. Clifford P. Fitch, assistant professor of bacteriology.
Dr. Earl M. Pickens, assistant professor in diagnosis.
Dr. S. A. Goldberg, assistant in pathology.
Mr. William A. Billings, assistant in diagnosis.
Mr. C. M. Carpenter, student assistant.
Mr. Herbert L. Gilman, student assistant.

Department of Obstetrics and Research in the Diseases of Breeding Animals

Dr. W. L. Williams, professor and head of the department.
Dr. W. A. Hagan, instructor.
Miss Ethel Williams, assistant.

Department of Surgery

Dr. J. N. Frost, assistant professor of veterinary surgery.
Dr. F. C. Clark, instructor.

Department of Physiology

Dr. Pierre A. Fish, professor of veterinary physiology and secretary of the faculty.
Dr. Chas. E. Hayden, assistant professor.

Veterinary Experiment Station

Dr. Raymond R. Birch, superintendent of the station.

Farriery

Mr. Henry Asmus, assistant professor of horseshoeing.

Library

Miss Frances B. van Zandt, librarian of the Roswell P. Flower Library.

Business Office

Miss Helena H. Haight, clerk and bookkeeper.
Miss Lulu M. Williams, stenographer and office secretary.

Engineers

Mr. Archibald Wilson, engineer.
Mr. Charles Savercool, assistant engineer.

Thirteen grooms, janitors and attendants were required to do the necessary work in the care of buildings, patients in the hospitals and experimental animals and help in the different departments and laboratories. In addition to these, special work necessitated for short periods of time the employment by the day or hour of a few men.

It is the practice to have during the year lectures from prominent practitioners or specialists in veterinary science or those engaged in some subject associated with the raising of live stock. This year the college was honored by helpful and instructive addresses from the following named persons. Some of the lectures were presented at the time of the conference.

Dr. John W. Adams, professor of veterinary surgery, University of Pennsylvania, Philadelphia, Pa.

Dr. A. R. Ward, Bureau of Animal Industry, United States Department of Agriculture, Washington, D. C.

Dr. J. W. Harshberger, professor of botany, University of Pennsylvania, Philadelphia, Pa.

Dr. George A. Knapp, practitioner of veterinary medicine, Millbrook, N. Y.

Dr. W. H. Kelly, practitioner of veterinary medicine, Albany, N. Y.

Dr. W. G. Hollingworth, practitioner of veterinary medicine, Utica, N. Y.

Dr. J. G. Wills, chief veterinarian, Department of Agriculture, Albany, N. Y.

Mr. C. J. Huson, breeder, Dresden, N. Y.

STUDENTS

As this was the beginning of the four year curriculum, it was expected that there would be a falling off in the number of new students. The reduction, however, was not great. For the five years prior to 1915 the average number of new students was 48. In 1916, the entering class numbered 36. It has been the experience in all colleges in the university, that when additional requirements were imposed there was a temporary dropping off in the number of students but very soon thereafter the number was larger than before. In 1905, when the Education Department exacted the full 72 regents' counts for a veterinary student certificate, instead of 24 as previously accepted, the number of students in the entering class dropped from 56 to 16. The following year there was an increase which continued annually until this further advance.

The conditions governing the attendance of undergraduates may be divided into three groups, namely: (1) from 1896 to 1905, during which time students could enter with 24 regents' counts; (2) from 1905 to 1915, when the entrance requirements were 72 regents counts and a three year course of instruction; and (3) from 1916 with the high school entrance and a four year course. The average annual registration of undergraduates during these periods was as follows:

Average number of students in attendance from 1896–1904 was 48.

Average number of students in attendance from 1905–1915 was 109.

The number of students in attendance in 1916 was 153.

It should be stated that the large entering class in 1915 makes the total registration for 1916 slightly larger than it probably will be for the next five years but not so large as the average is likely to be for the first ten years of the four year course. These figures are indicative of the approval that has been given to the steady advance in higher veterinary education.

During the first twenty-one years the college graduated 439 students. There were 153 who entered but did not complete their course. Of the 439 graduates, 385 or 87 per cent came from New York State; 47 came from 16 states other than New York; and 7 came from six foreign countries. A fact significant of the orientation of interest in veterinary medicine, is the small number of the students who came from large cities and the large number that came from towns and rural districts. It is gratifying to report that, after twenty years, this college with its full university requirements for both entrance and graduation can supply the State with all the veterinarians its live stock possessions require.

Alumni

This year 32 students received their degrees. Fifteen of them took and passed the examination for officers in the veterinary corps of the regular army and practically all of the others took the examination for the veterinary officers' reserve. It was the intention of the majority of these men to practice in New York State prior to the declaration of war.

It is of interest to know the occupation of the 407 alumni who graduated prior to 1917. Those who do not believe veterinary education is a State function have raised the question whether men receiving technical training in such institutions subsequently use their knowledge for the benefit of the State. The college has kept as complete records of its graduates as possible and from these the following data have been gathered. There are, in private practice, 226; as veterinary inspectors in the federal government, 54; teaching, 36; in laboratory work, 20; in dairy inspection, 19; army veterinary officers, 9; in veterinary administrative work, 5; professors of animal or dairy husbandry, 2; unknown, 8. Eleven have gone into other business and 17 have died. *Of the 390 living, 246 are in New York State.*

There were certain important factors that influenced the occupation of many of the earlier graduates. The State already had a large number of established practitioners. The United States Bureau of Animal Industry was in great need of veterinary inspectors and the agricultural colleges and experiment stations were in search of teachers of veterinary subjects and men to do research on animal diseases. Because of these demands, many of the older alumni accepted places with the federal government and in educational institutions. During the last eight years, however, most of the graduates have taken up practice, and with few exceptions, in New York State.

A careful inquiry into the veterinary needs of the State indicates that it requires from 25 to 30 new veterinarians annually.

INSTRUCTION

The purpose of the college, as a teaching institution, is to provide instruction necessary to make efficient practitioners. This calls for careful selection of subject-matter from the basic sciences and opportunity for teaching the application of this knowledge in the practical subjects of medicine, sanitary science and surgery. The rapid acquisition of new knowledge renders the selection of the most important topics in many of the sciences a difficult task. For this reason an entirely satisfactory curriculum is impossible. However, the several departments are adjusting their work to meet these advances.

The following curriculum was rigidly observed. It consists of the first year in the four year course and the second and third years of the three year course.

PRESCRIBED WORK FIRST YEAR, FOUR YEAR COURSE

First year	Number of course	Credit first term	Credit second term	Total of actual hours
Physics	2	5	.....	75
Histology	6	3	3	60
Anatomy	1	3	.....	405
Anatomy	2	1	.....	
Anatomy	3	2	.....	
Anatomy	4	.....	6	
Physiology, recitations	11	.....	3	90
Physiology, lectures	10	3	.....	
Chemistry	1	.....	6	135
Military drill	1	.....	.....	90
		17	18	855

PRESCRIBED WORK LAST TWO YEARS OF THE THREE YEAR CURRICULUM

Second year	Number of course	Credit first term	Credit second term	Total of actual hours
Anatomy	5	4	.....	225
Anatomy	6	2	.....	
Physiology	11	1	.....	15
Physiology	13	1	.....	15
Pharmacology	20	2	.....	30
Materia medica and pharmacy	21	2	.....	75
General pathology	40	4	.....	105
Special pathology	41	.....	2	53
Parasites	44	2	.....	53
Small animal clinic	25	.....	1	45
Consulting clinic	53	.....	1	45
Bacteriology	43	.....	5	143

Second year	Number of course	Credit first term	Credit second term	Total of actual hours
General surgery	30	.....	4	97
Physical diagnosis	51	.....	2	30
Horseshoeing	52	.....	1	15
Ophthalmology	55	.....	1	15
Hygiene	56	1	.....	15
Horseshoeing exercises	57	.....	1	45
		19	18	1,021

Third year	Number of course	Credit first term	Credit second term	Total of actual hours
Embryology	....	.....	1	54
Urine analysis	15	1	.....	45
Diseases of small animals	22	.....	2	30
Materia medica and therapeutics	23	2	.....	30
Surgical exercises	31	1	.....	45
Special surgery	32	4	.....	60
Obstetrics	36	.....	4	60
Infectious diseases	42	.....	2	30
Special pathology	41	2	.....	53
Meat and dairy inspection	48	.....	1	15
Small animal clinic	25	1	1	90
Consulting and medical clinic	53	1	1	90
Surgical clinic	34	2	2	180
Ambulatory clinic	37	1	1	90
Medicine	50	5	5	150
		20	20	1,022

During the year much has been done by formulating methods that will intensify instruction and aid the student in an understanding of the different subjects. Instructive charts have been made from actual dissections for illustrating the exact location of

the various organs with reference to the surface of the body for use in teaching physical examination and diagnosis. Additions have been made in the equipment and in the facilities for teaching horseshoeing. Many accessions of teaching value have been made to the pathological collection.

The teaching of chemistry, embryology, histology and physics is in the respective departments in the university. The laboratories of anatomy, bacteriology, physiology and pathology in this college are well appointed and the teaching is by experienced men. In the department of medicine, provision has been made for all the necessary tests in connection with the clinical examinations and diagnosis work. The clinics have improved much this year. As the college is surrounded by a farming community it is enabled to maintain varied and most instructive ambulatory and consulting clinics. The small animal clinic is well patronized.

A study of the detailed reports of the consulting and surgical clinics, as given in the appendix, will reveal the variety of diseases treated and the opportunities available for giving instruction in operative surgery. Animals are sent to this hospital from long distances for especially delicate or difficult operations. The same conditions hold for the small animal clinic.

The ambulatory clinic has demonstrated the economic significance of prompt and efficient veterinary service. It has shown that with early attention and careful watching of cases until full recovery, many animals which otherwise would be lost are restored to usefulness. This clinic has demonstrated the important part veterinarians can take in the conservation of the live stock of the country when they have the confidence and co-operation of the animal owners. It is significant that in this clinic are many cases of sterility and abortion in cattle. This affords exceptional opportunity for students to become fully conversant with the methods of treating these disorders and to compare herds which have been carefully treated, where the number of cases has come to be very small, with those that are treated indifferently or not at all.

The appended table gives the data concerning the number of cases treated and shows the steady improvement in facilities for the practical teaching of medicine and surgery.

CLINICS

	Average number of cases treated annually for 8 years 1907-14.	Number of cases for 9 mo. Oct 1, 1915, to July 1, 1916.	Number of cases treated in year ending June 30, 1917.
Ambulatory	887	1,499	2,694
Consulting	302	451	578
Surgical	184	175	182
Small animal	338	397	483
Total	1,711	2,522	3,937

The teaching value of the clinics is not entirely determined by the number of cases. The real value is in the opportunity that students have to study the cases until recovery or death and, if the latter, to have a careful post mortem examination. A room for this purpose has been equipped and all fatal cases are carefully examined. Histological and bacteriological examinations are made of the different organs. This enables the student to check the clinical diagnosis with the post mortem determinations. These examinations are made by the department of pathology where they are utilized in teaching gross pathology. They are also of much assistance in collecting data on general comparative pathology.

There is much teaching value in the specimens that are sent to the college for diagnosis as they often furnish material for demonstrating lesions of certain diseases that otherwise could not be obtained. An optional course in laboratory diagnosis is offered to senior students in which they have an opportunity of doing under supervision the work required to determine the nature of the disease. This prepares them to make positive determinations that would take valuable time if the specimens were sent away. This is important, for a quick diagnosis often enables the veterinarian to check the spread of destructive epizootics.

An important practical course is offered in surgical anatomy. This is designed especially to emphasize the details of the anatomy of the regions of the body most subject to surgical operations. This course is available not only for students but also for practitioners who wish to advance their surgical work.

Library

The library contains 5,359 volumes of which 5,052 belong to the Flower Library and 307 to the State. There were added during the year 331 volumes. The library received 96 periodicals on veterinary medicine and closely allied subjects, human medicine and a few journals on animal breeding. It is in charge of a trained librarian who also instructs the students in methods of cataloging books and in using a library.

We have the privilege of announcing the gift from Dr. L. McLean of Brooklyn, N. Y., of his private veterinary library of 61 volumes. Professor S. Jacoby presented the library with the proceedings of the Sixth International Congress on Tuberculosis. The college is very grateful for such gifts.

Laboratory Diagnosis

The diagnosis of specimens of diseased tissues and organs for veterinarians of the State and for the Commissioner of Agriculture has continued as in previous years. There was a much greater demand for diagnostic agents and anthrax vaccine than heretofore. The appended table gives a list of specimens examined and the biologic products made during the year ending June 30, 1917.

Examinations Made and the Amount of Tuberculin, Mallein and Anthrax Vaccine Sent Out During the Year Ending June 30, 1917

Examinations for anthrax	70
Examinations for blackleg	18
Examinations for glanders (agglutination)	166
Examinations for hog cholera	10
Examinations for mastitis in cattle	41
Examinations for poultry diseases	404
Examinations for rabies	190
Examinations for tumors	45
Examinations for tuberculosis	182
Examinations, miscellaneous	238
Anthrax vaccine sent out (doses)	18,251

Mallein sent out (doses)	673
Tuberculin (old) sent out (doses)	76,756
Retest tuberculin (doses)	27
Precipitated tuberculin (doses)	2,280

Under miscellaneous a large variety of morbid specimens were examined. Some of these, like Johne's disease and swamp fever, are significant in pointing to the existence of these diseases within the State. The poultry specimens included infectious enterohepatitis, pseudoleukaemia and tuberculosis with a large collection of the more common maladies. The tumors included fourteen carcinomas and five sarcomas. The others were largely adenomas, fibromas and papillomas.

Anthrax.— Of the seventy specimens examined for anthrax, twenty-nine were positive and forty-one were negative. The positive ones came from twenty-four localities which is a much larger number of infected areas than was found last year. Attention has been called in previous reports to the importance of making an early diagnosis of this disease and instituting prompt measures to prevent its further spread. The persistence of the anthrax spores renders it necessary to prevent as much as possible soil contamination from sick or dead animals. More anthrax vaccine was called for than during any previous year.

SPECIMENS OF SUSPECTED ANTHRAX RECEIVED FOR EXAMINATION DURING THE YEAR ENDING JUNE 30, 1917

MONTHS	Positive	Negative	Total
1916			
July	5	10	15
August	6	6	12
September	3	4	7
October	3	3	6
November	1	2	3
December	.....	3	3
1917			
January	1	1	2
February	.....	.....	.....
March	.....	1	1
April	3	5	8
May	3	1	4
June	4	5	9
Totals	29	41	70

ANTHRAX VACCINE SENT TO VETERINARIANS AND TO THE COMMISSIONER OF AGRICULTURE DURING THE YEAR ENDING JUNE 30, 1917

MONTHS	PRIVATE		STATE	
	No. I	No. II	No. I	No. II
1916				
July	105	105	800	500
August	860	880	1,548	2,048
September	90	90	500	500
October	200	100	.....	.....
November	.....	.....	.....	.....
December	.....	.....	5	5
1917				
January	.....	.....	.....	.....
February	.....	.....	.....	.....
March	.....	.....	500	500
April	775	775	2,000	2,000
May	450	415	1,000	1,000
June	280	220	.....	.....
Totals	2,760	2,585	6,353	6,553

Glanders.— There was a reduction in the number of samples of blood for the agglutination test for glanders and also in the amount of mallein called for.

AGGLUTINATION TEST FOR GLANDERS DURING THE YEAR ENDING JUNE 30, 1917

MONTHS	500	800	1000	Negative	Impossible	Total
1916						
July	2	4	6	7	.....	19
August	.....	.....	6	1	.....	7
September	1	1	3	2	1	8
October	5	1	2	1	.....	9
November	1	.....	3	1	.....	5
December	3	.....	2	5	.....	10
1917						
January	3	.....	2	1	.....	6
February	4	.....	.....	1	.....	5
March	2	.....	.....	2	.....	4
April	18	3	13	37	.....	71
May	3	.....	.....	7	.....	10
June	.....	.....	7	5	.....	12
Totals	42	9	44	70	1	166

NUMBER OF DOSES OF MALLEIN DISTRIBUTED IN THE STATE DURING THE YEAR ENDING JUNE 30, 1917

MONTHS	Private for subcutaneous test	For eye test	State for subcutaneous test	Total
1916				
July	20		200	220
August		10		10
September	12			12
October		20	100	120
November		10	100	110
December	5			5
1917				
January		10		10
February			150	150
March				
April	6			6
May	10	10		20
June	10			10
Totals	63	60	550	673

Rabies.— There was an increase of nearly 75 per cent over the previous year in the number of specimens from animals suspected of having this disease. Of the 190 specimens received, 86 were positive, 92 were negative and 12 could not be determined because of the condition they were in when received. As heretofore, there were a few specimens from animals other than dogs. The losses among cattle, sheep and horses from rabies during the last years, emphasize the necessity of veterinarians being very careful to detect this disease and to prevent exposure of food producing animals. The unofficial statement was made recently that in one county over $1,000 worth of animals died of rabies in one year. The source of infection in all these cases was the bites of rabid dogs.

TABLE SHOWING BY MONTHS THE NUMBER OF SPECIMENS RECEIVED FOR EXAMINATION FOR RABIES DURING THE YEAR ENDING JUNE 30, 1917

MONTHS	POSITIVE				NEGATIVE				IMPOSSIBLE		
	Dog	Cat	Cow	Sheep	Dog	Cat	Cow	Horse	Dog	Cow	Total
1916											
July	6	1	.....	.....	6	1	1	.....	2	1	18
August	3	.....	.....	.....	7	.....	2	.....	2	.....	14
September	3	.....	1	.....	3	.....	.....	.....	2	.....	9
October	6	.....	.....	.....	4	.....	1	1	1	.....	13
November	8	.....	.....	.....	1	.....	.....	.....	.....	.....	9
December	15	.....	.....	.....	4	.....	.....	.....	.....	.....	19
1917											
January	13	.....	.....	.....	8	.....	3	.....	.....	.....	24
February	5	.....	1	.....	7	.....	2	.....	1	.....	16
March	7	.....	.....	.....	9	.....	.....	.....	.....	.....	16
April	5	.....	.....	.....	9	.....	.....	.....	.....	.....	14
May	5	.....	3	1	7	.....	1	.....	2	.....	19
June	2	.....	.....	1	15	.....	.....	.....	1	.....	19
Totals	78	1	5	2	80	1	10	1	11	1	190

Tuberculosis.— There were examined 182 specimens from animals suspected of having tuberculosis. Last year 103 such specimens were received. Of the 182, only 97 were positive. The large number of specimens that were not tuberculous illustrates the frequency of the occurrence of tissue changes that are easily mistaken for lesions of this disease. There has been a steady increase in the call for tuberculin. The demand for precipitated tuberculin for intradermal use was negligible.

SPECIMENS OF SUSPECTED TUBERCULOSIS RECEIVED FOR DIAGNOSIS DURING THE YEAR ENDING JUNE 30, 1917

MONTHS	Positive	Negative	Total
1916			
July	9	2	11
August	3	5	8
September	1	2	3
October	8	4	12
November	12	23	35
December	2	5	7
1917			
January	39	8	47
February	.....	2	2
March	10	23	33
April	3	2	5
May	8	6	14
June	2	3	5
Totals	97	85	182

TUBERCULIN DISTRIBUTED DURING THE YEAR ENDING JUNE 30, 1917

MONTHS	REGULAR		RETEST		PRECIPITATED		Total doses
	Private	State	Private	State	Private	State	
1916							
July	1,563	1,500	5	.....	20	.....	3,088
August	2,574	1,500	.....	.....	20	.....	4,094
September	2,816	3,000	.....	.....	490	.....	6,306
October	3,181	6,600	10	.....	20	.....	9,811
November	3,433	3,325	.....	.....	20	.....	6,778
December	3,680	3,430	2	.....	.....	50	7,162
1917							
January	3,020	2,300	10	.....	30	1,260	6,620
February	2,765	2,500	.....	.....	10	.....	5,275
March	4,143	4,100	.....	.....	70	20	8,333
April	4,034	3,000	.....	.....	210	.....	7,244
May	3,787	3,250	.....	.....	10	.....	7,047
June	2,180	3,600	.....	.....	50	.....	5,830
Totals	37,176	38,105	27	.....	950	1,330	77,588
Returned	.....	.....	.....	.....	.....	.....	834

Swine Diseases.— Hog cholera is the most important infectious disease of swine in this State. In the report for 1915–16 a detailed description of the method for its control by the use of anti-hog-cholera serum was given. In the same report were recorded the results of experiments, which showed that the virus of this disease is carried in the flesh and bones of infected hogs that are killed before lesions have developed sufficiently to enable the diagnosis to be made.

Much help has been given by the college to veterinarians who have encountered difficulties in checking this disease. Emphasis should be placed on the importance of an accurate diagnosis and of following the instructions for the use of the serum alone and the simultaneous method. The certainty with which hog cholera can be controlled makes it possible to feed garbage with profit and thereby convert into meat, refuse that is too often wasted.

During the year ending June 30, 1917, there were sent out from this college 254,360 c.c. or 12,718—20 c.c. doses of anti-hog-cholera serum. This was an increase over any previous year. The results from its use have been generally satisfactory. The simultaneous method is used when it is necessary. As there is no quicker way of increasing our meat food and fats than by growing swine, it is important that every possible precaution be taken to

control hog cholera. In this, local veterinarians have an important work first in making a correct diagnosis and secondly in properly using the serum or the serum and virus.

Poultry Diseases.— There were received 404 specimens for diagnosis from domestic fowls. This was an increase of nearly 50 per cent over the previous year. The losses from diseases of poultry are increasing and their economic importance is becoming more and more serious. Veterinarians must pay more attention to these disorders or non-professional men, who have studied the diseases of poultry only, will appear to look after them. This would be unfortunate. We have only to look to human medicine to find men who have been trained in one or two elements in the treatment of disease clamoring for a legal right to practice medicine. It is hoped the veterinary profession will be saved from similar experiences.

The diseases which at present are causing the greatest loss are chicken cholera, blackhead or infectious entero-hepatitis, bacillary white diarrhea, roup or diphtheria, chicken pox and the parasitic infestations. The knowledge of these affections is such, that practioners should be able to assist poultrymen in preventing or controlling them. The very serious losses from bacillary white diarrhea are causing poultry owners to appeal to veterinarians for assistance. As there is no other infectious disease that is more amenable to proper precautionary measures than this, it is recommended generally, and especially now, that more direct efforts be made by veterinarians to assist those engaged in this important branch of animal industry. The losses among poultry amount to hundreds of thousands of dollars annually in this State alone.

Research

The study of animal diseases has progressed with quite satisfactory results. In previous reports the plans for research work followed by the college have been explained. At this time, a number of investigations can be reported. Others are in progress and in due time will be published.

During the year a number of valuable papers have been published by various members of the faculty. Among these should be mentioned a most helpful article by Dr. Pickens on " Some of the

Infectious Diseases of Poultry." This was for the purpose of aiding veterinarians in preventing, diagnosing and treating these affections, which have come to be a serious problem in the State. In 1915, the farm census of live stock recorded over 17,000,000 fowls in New York. An important paper was published by Dr. Frost on "Formalin in the Treatment of Mastitis." This was the outcome of continued efforts to treat this trouble with chemicals that are eliminated largely through the udder. A third publication on the "Parasites of Sheep" by Dr. Fitch was made in advance of the report because of the revival of interest in sheep raising in the State. Dr. Fitch was making a careful study of these parasites when he resigned. The reports of the work on formalin in mastitis and the parasites of sheep will be found in the appendix.

Investigations have been in progress to determine, if possible, the relation between tuberculous lesions and the reaction to tuberculin. The several methods now in vogue for applying tuberculin render it desirable to ascertain which of them has the best claim for accuracy. It is hoped that in another year the results may be ready for publication. One of the theories put forth is that the reaction depends upon the antibodies given off from the tissues actually infected and which remain closely associated with the lesion. The sensitizing theory is apparently the basis for the intradermal test which experimentally has not been so accurate with us as the subcutaneous method.

An experiment has been started to ascertain if possible the immunizing effect of the bacterial protein of the Bang organism. This experiment was begun with young calves. It may require from two to three years to bring this to a conclusion. The economic importance of infectious abortion and the extent to which the bacterins for that trouble are advertised, made it desirable to give this method a thorough test. If it is of value the stockmen should know it and if it is not, that fact should be understood.

The appendix contains a detailed report of the clinics. This will indicate the large variety of diseases that are treated and the opportunity for students to receive instruction in the practical subjects. It also contains the results of a number of investigations.

The report of Dr. Pickens on tuberculosis in the horse is important in showing that in this country, as well as in Denmark, this disease exists among horses.

The work of Dr. Birch on garbage feeding, and the care of garbage fed swine, shows to what extent the veterinarian may be of value in the conservation of swine fed upon this waste. In this connection the attention of the veterinarian is called to the papers by Dr. Birch in the report of 1915–16.

The reports from the department of obstetrics and the diseases of breeding cattle are worthy of careful study. Throughout the world, wherever dairying is important, a group of chronic infections has become widely prevalent, causing serious losses in the form of sterility, abortion, retained afterbirth, calf scours, and allied forms of disease. Because of the decreased production caused by these diseases, the cost of each pound of milk, butter or cheese is stated to be increased more than ten per cent.

The importance of this group of diseases was early recognized by this college, and members of its faculty became pioneer investigators in this field. In recent years, the research and clinical work in this group of diseases has grown enormously. A study of the report of the Ambulatory Clinic will show that sterility and retained afterbirth have become of first importance among diseases of cattle. Numerous sterile cows are entered in the Surgical and Consulting Clinic for treatment. Further, some of the most valuable cows from this and other states come to the Department of Research in the Diseases of Breeding Animals, for treatment for sterility.

From the small beginnings of casual study in the clinic, the research work has developed into a department with one professor and one instructor devoting essentially all their time to this subject, in addition to the work carried out in other departments. A suitable building has been erected for the new department, with good accommodations for thirty cattle. The value of the work is increasing rapidly and the department is striving to anticipate the growing importance of these diseases and the increasing demand for more knowledge of this subject by veterinarians, cattle breeders and dairymen.

The report on genital tuberculosis in cattle by Dr. Williams

is one of the most comprehensive articles on this subject. It contains much of value to the clinician.

The report by Dr. Hagan on the etiology and mode of infection in white scours of calves is a valuable contribution. The group of somewhat chronic affections associated with white scours such as joint lesions and broncho-pneumonia are clearly set forth in this report. Another interesting observation was the bacterial content of the meconium which indicates that, contrary to general opinion, the uterine cavity frequently contains bacteria other than the Bang organism.

The researches of Dr. Goldberg on the lesions in spavin are illuminating. The changes in the bone and articular cartilages, which he found to characterize this disease, suggest infection as the etiological factor.

Important among the investigations are those of Dr. Muldoon on anæsthesia. The elimination of pain in surgery is of great value as it makes possible many operations that otherwise owners would not permit. It has been the practice in the surgical clinic of this college to use anæsthetics. There are, however, many difficulties involved which the results of the careful work of Dr. Muldoon will remove. This paper will be of much assistance to veterinary surgeons. It will be appreciated also by many people who, for economic reasons, desire to have operations on their animals but refrain because of the supposed suffering. This investigation will do much to humanize veterinary surgery.

The study of the parasites of the dog by Dr. Dederick has brought out useful data relative to the diagnosis of parasitic troubles by microscopic examinations of the feces. The frequency of hookworms is of special interest.

The report of Dr. Hayden on "Orokinase and Ptyalin in the Saliva of the Horse" is a valuable contribution to the important discussion of the digestive action of the salivary and mouth secretions of the horse. "Orokinase" is the name applied by a recent worker to an enzyme supposed to be secreted in the mouth of the horse. The enzyme is said to activate the saliva secreted by the main salivary glands of the horse. The activation results in a ready digestion of starch. The results obtained by Dr. Hayden's investigations have led to the conclusion that saliva as well as the

secretions of the glands and mucous membranes of the mouth of the horse have a slight digestive action on starch but the presence of the new enzyme is not confirmed.

Outside Educational Work

The purpose of this college is not only to teach undergraduates but also to give practitioners every possible assistance that may contribute to their efficiency. Veterinary medicine is a growing profession and every year there are accessions of new knowledge and new methods of control or treatment that should be disseminated among the veterinarians who are occupied with the details of practice. It is essential, therefore, that the State college should be an institution to which the practitioners of the State can go for assistance and also an agent through which new knowledge concerning the diseases of animals can be disseminated promptly to those who are to apply it for the conservation of animal life. The efforts of the college along these lines have brought about better cooperation between the practitioners and teachers. Among the special efforts to make this institution valuable to the veterinary service of the State the following activities may be mentioned, namely:

Conference for Veterinarians.— This was held January 16 and 17 and was largely attended by the practitioners of the State. As previously pointed out, these gatherings are for the purpose of having the newer developments in veterinary medicine presented to and discussed by the veterinarians. On this occasion a symposium on the poisoning of animals was most helpful. Other important subjects were considered. A most helpful feature of these gatherings is the informal discussions between the practitioners themselves. These bring out the veterinary problems in the different localities and the special efforts that are necessary to solve them. The success of the conference is assured from its increasingly large attendance.

Fair.— The college co-operated with the State Food Supply Commission in preparing its exhibit for the State fair. By means of photographs, diagrams and charts, it presented the ways by which bovine tuberculosis is spread and how it can be prevented, or if present, eradicated. The exhibit was attended by

men of the faculty who could answer questions and explain in more detail the subject of disease control. The exhibit attracted much attention and many questions were asked. It is believed that some good accrues from these exhibits and their explanation.

Correspondence.— This is one of the agencies for aiding veterinarians and live stock owners. There are also many requests for the college publications and for assistance in various other ways. These consume considerable time but it is believed that they are a positive help to both practitioners and animal owners.

Miscellaneous.—A large amount of time is occupied by various members of the faculty in assisting veterinarians and in discussing problems involving the control of animal diseases at different live stock and veterinary organizations. The effort here is two fold; first, to have animal owners come to understand what the veterinarians are able to do for them and secondly, to give such aid as may be requested by the practitioners in performing their part. The efficiency of the veterinary profession in both preventing and treating animal diseases depends largely upon the initiation of the animal owners and the promptness with which they seek assistance after disease appears.

In case of hog cholera, it occasionally happens that both the owner and his veterinarian become dissatisfied with the results. The proper assistance given on the technical side of the subject has enabled the owner to save his animals and the veterinarian to demonstrate his worth. It is frequently a necessity that more or less assistance be given for the successful application of preventive, operative or therapeutic measures. It is believed that a prompt response to calls for assistance in these matters is most beneficial. It is unfortunate that more cannot be done in this direction.

NEEDS OF THE COLLEGE

The need for buildings is the same as it has been for three years. The wing to the main building for auditorium, library purposes and offices is still wanting. A building for the State diagnosis work, the preparation of diagnostic agents and vaccines, the laboratories for bacteriology, pathology, post-mortem and parasitology, and for research laboratories for toxicology and chemistry is badly needed. The use of the same laboratories

for teaching undergraduates, for research and the diagnostic work is most unfortunate. This work has outgrown its facilities.

The enormous losses from diseases of animals have been recognized by the Federal government and coincident with the declaration of war were large appropriations by Congress to combat animal diseases. This money is being spent in cooperation with various states. The prevalence in this State of hog cholera, tuberculosis, infectious abortion, anthrax and poultry diseases warrant the appropriation of funds sufficient to cooperate with the government in preventing these losses.

It should be recognized that there are many facts yet to be ascertained concerning the nature of these maladies before successful regulatory measures can be put in operation. This means that there should be well directed research work carried on in addition to the more striking field demonstrations. These studies on the fundamental problems are of vital importance in formulating preventive methods. For this reason it is urgently recommended that the appropriation for this work be increased.

Recommendations

It is recommended that the trustees ask the Legislature for the following appropriations for the fiscal year 1918–19:

For maintenance, including research work.....	$80,000 00
For repairs, new roof, main building.....	900 00
For the south wing to the main building.....	150,000 00

FINANCIAL STATEMENT

REPORT OF THE CIRCULATING FUNDS OF THE NEW YORK STATE VETERINARY COLLEGE AT CORNELL UNIVERSITY JULY 1, 1916 TO JUNE 30, 1917

Balance June 30, 1916		$10,657 17
Receipts July 1, 1916 to June 30, 1917:		
Tuition	$1,865 00	
Laboratory fees	3,732 92	
Clinics and medicine	5,259 09	
Tuberculin and mallein	2,286 48	
Hog cholera serum	2,317 19	
Animals and animal products	1,095 18	
Horseshoeing	1,396 79	
Miscellaneous	231 20	
		18,183 85
Disbursements July 1, 1916 to June 30, 1917:		
Laboratory supplies	3,628 42	
Clinics and medicine	5,384 51	
Tuberculin and mallein supplies	2,078 84	
Serum supplies	2,334 38	
Farriery supplies	1,201 54	
Teaching supplies	503 04	
Research supplies	1,410 50	
Extension work supplies	954 19	
Fuel, light, power and water	853 55	
Office and library supplies	490 71	
Minor repairs	215 41	
Minor improvements and equipment	633 97	
Labor	685 00	
Non-resident lecturers	78 95	
Miscellaneous	628 60	
		21,081 52
Balance June 30, 1917		7,759 50
		$28,841 02

Expenditure of Maintenance Fund for the New York State Veterinary College at Cornell University for the Year 1916–1917

(Chapter 646, Laws of 1916, Part 1)

Personal service	$57,623 46
Fuel, light, power and water	1,821 26
Printing	599 72
Advertising	48 85
Equipment	1,037 83
Supplies	5,092 84
Materials	1,070 68
Traveling expenses	99 74
Communication	661 03
General plant service	97 18
Contingencies	67 35
Total spent	68,219 94
Balance lapsing to State	1,370 06
	$69,590 00

Expenditures for Equipment of Clinical Buildings for the College Year 1916–1917

(Chapter 646, Laws of 1916, Part 3)

Expended July 1, 1916 to June 30, 1917:		
Equipment of clinical buildings		$2,084 41
Heat and partitions in farriery		230 29
		2,314 70
Balances June 30, 1917:		
Equipment clinical buildings	$5,415 59	
Farriery	1,269 71	
		6,685 30
		$9,000 00

Expenditures for Special Investigation of Diseases of Breeding Cattle

(Chapter 629, Laws of 1916)

Expended July 1, 1916 to June 30, 1917.....	$1,701 11
Balance on hand June 30, 1917.....	13,298 89
	$15,000 00

As per itemized vouchers approved by the State Comptroller.

Respectfully submitted,

H. H. Haight,
Clerk.

In the appendix will be found the detailed reports of the clinics and researches which should be regarded as an integral part of this report.

In carrying out the requirements of the law governing the work of this college, the members of the faculty have worked unitedly to the end that the college shall meet the needs of the veterinarians and live stock owners of the State and also fulfill its obligations to the students.

Respectfully submitted,

V. A. MOORE,
Dean, New York State Veterinary College at Cornell University, Ithaca, N. Y.

APPENDIX

Containing Reports of Clinics and Research Work

REPORT OF THE AMBULATORY CLINIC FROM JULY 1, 1916 TO JUNE 30, 1917

D. H. Udall, F. F. Koenig, J. B. Latshaw

Department of Medicine and Ambulatory Clinic

During the past year the ambulatory clinic has been prosperous in respect to the number of cases treated, and the completeness of the individual records. For the first time reliable and comprehensive data concerning the termination of cases have been fairly complete. This information is sometimes difficult to obtain due to the exchange of animals and the amount of effort required to identify and follow them up.

The increase in numbers is gratifying; for the first time it includes none of the animals from the Department of Animal Husbandry. These records are omitted since they are too incomplete and fragmentary to be of value. It should also be noted that they do not include the usual annual tuberculin test of more than 100 animals. Regardless of this deflection there is a gratifying increase in animals treated.

Because of the importance attached to certain infectious diseases special attention is called to their solution from the standpoint of a clinician, a view-point that is one of the many assets of the ambulatory clinic.

Of the various diseases listed as infectious, *contagious abortion* is in our experience of chief economic importance. Its significance is not limited to the 19 cases listed among cows; many cows listed as sterile have an unrecorded history of abortion, and had they been treated then there would have been no sterility. An examination of our case records shows that practically every animal given thorough treatment at the time of abortion has conceived; several have since delivered live calves, and others appear safely pregnant. It appears that the successful treatment of abortion consists in curing all metritis as soon as possible after the

fetus is expelled, and that the chances for a subsequent conception and delivery are in direct proportion to the thoroughness of this operation. According to our experience the number of treatments essential to a recovery are seldom more than is warranted by the value of an ordinary grade cow; they should be started as soon as the fetus is expelled and continued until there is no pus or other evidence of metritis.

The solution of the disease is somewhat complicated, perhaps more so to a clinician than to any other person, since he comes in personal contact with all of its angles. Owners are often reluctant to accept a diagnosis of abortion when the fetus is delivered at term, so that some of the most dangerous cases may escape treatment. Unless one accepts the other extreme of considering all metritis as evidence of abortion the diagnosis often presents real difficulties. Owners sometimes sell animals that have aborted, thinking that the disease is incurable and that once abortion occurs the cow is ruined. This is unfortunate for several reasons: the cow is sold and is either butchered or spreads the contagion through the herd of the purchaser (contamination of the bull, milk, and possibly other non-pregnant cows), in either case she escapes the treatment that might have returned her to useful breeding. In some cases an aborting animal seems to have an infection so mild in character that the metritis clears up without treatment and she conceives in a few weeks. It would be much safer for the aborting cow and for others in the herd if such animals could have uterine douches to insure conception in a thoroughly cured uterus. This mild group of cases seems to benefit greatly from a single thorough irrigation of the uterus. In a recently treated case of abortion at seven months the uterus was found normal and contained the normal glassy secretion of estrum at the end of three weeks. The misconception among certain dairymen that abortion is incurable should be corrected. They should be made to realize that cases of metritis following abortion, or due to any other condition, are not only ruinous to the infected individual but a menace to the entire herd; a harvest that may not be reaped until after the original cow is forgotten.

Fortunately a herd control of the disease whereby practically all of the cows produce live, healthy calves does not depend on a

settlement of this or any other controversy. As clinicians develop skill and enterprise in the treatment of metritis they will come to have complete control in the herds of their better clients and both will be gratified with the results. The value of State and Federal control of this infection through the dissemination of information should not be underestimated, at the same time it must be recognized that once abortion has taken place in a cow only nature or a skilled hand, or both, return her to the herd in a healthy condition. From the standpoint of the clinician, then, the only "cure" is in his hands and it is the duty of the State to produce the type of clinician fitted for the work, and no other. The disease cannot be cured through bulletins, or lectures, or even demonstration; irrigation of the uterus demands continuous experience.

The relation between milk from infected cows and abortion is sometimes so striking as to impress even the owner. One client recently called attention to two unthrifty heifers both of which became pregnant when too young, and both aborted. In company with a young bull by which they were served they were raised on the mixed milk of a small herd containing a persistent aborter; they have never mingled with any other animals and the only visible source of contact is through the milk from this cow. Of seven animals sired by this young bull, five aborted.

General principles of prevention are best applied through cooperation between the owner and the clinicians. Under herd conditions in the average dairy district the amount of new infection is largely a matter of luck. Since these conditions have a direct bearing on the cost of production of dairy products there should be some systematic and intelligent control by men of experience. Some of our leading authorities on the cost of milk production speak from experience in herds where nature takes her toll with little interference.

It is not within the scope of these comments on our experience with the treatment of the disease to discuss principles of prevention. Success in suppressing the infection is seldom absolute; in nearly every herd cases reappear from time to time. No single operation is capable of preventing its recurrence. Continuous vigilance on the part of the dairyman and his veterinarian seems to offer the most satisfactory solution of the problem.

Since the method of treatment of metritis in all its forms is a development of the last few years men in practice who graduated a short time ago are often without the skill essential to success. It is within less than ten years that clinics for teaching this technique have been etablished. Unlike other departments in the veterinary college they have been either self-supporting or a source of profit to the institution. At the present time no phase of clinical teaching is of greater importance or merits greater support. I believe that the teaching of cattle diseases in the ambulatory clinic offers one of our greatest opportunities for the support of animal industry and the development of the veterinary profession.

To meet the possible needs of men in practice I would recommend that special courses be given at the college on the treatment of cattle diseases, such courses to be given to men now in practice who feel the need of instruction that was not given in their day.

Tuberculosis is one of the infectious diseases in animals in which the State assumes complete responsibility, and it is the one that is apparently making greatest progress among our clients. In the ambulatory clinic our connection with the case ends when it is diagnosed and reported. In some cases the tuberculous animal is destroyed by the owner, in others he requests aid from the State and calls for a test. So far as we know in this county the final result is the same; sooner or later the herd reverts to its original condition until we find another tuberculous animal, when the process is repeated. Given the same freedom for cooperation with our clients that we have in other contagious diseases I believe we would soon be certain that a few herds in the county were not only free but under a supervision that would keep them free. Under our present system suspected animals are often sold without the owner knowing that they are tuberculous, while it is inevitable that certain others are open cases of the disease and are not even suspected.

Individual cases of important infectious diseases have been found during the year; namely, anthrax, Johne's disease, and blackleg. The numbers are small but their presence even is significant of the distribution, and a possible future spread. So far

as we know these are the first reported cases of Johne's disease and anthrax in Tompkins county herds.

Another condition of great economic importance among cows is that of *retained placenta.* In the ambulatory clinic report of last year the opinion is expressed that abortion, metritis, and retained placenta are practically identical in meaning, that metritis or retained placenta are complete evidence of infection with the abortion bacillus of Bang. Further experience has shown that evidence of such an association is wanting. While it is true that some of our worst cases of acute metritis are those associated with retained placenta of this specific abortion infection, it does not yet seem logical to consider all, or nearly all, cases of retained placenta as complete evidence of such infection. Regardless of any theory concerning the etiology it is a very frequent and obvious condition that through neglect and improper treatment is the source of great economic loss. In the ambulatory clinic we have been trying various methods of treatment and with experience obtained from the handling of many cases are gradually obtaining satisfactory results in practically all cases. As in the treatment of most diseases it cannot be expressed in exact formulae. It is only by experience and skill combined with a certain amount of intuition that one learns to judge the best treatment for each case. In the past year about 60 have been treated; in point of frequency it shares the head of the list with sterility.

Each year there is an increased demand for the treatment of *sterility* and in herds where the treatment has been continued for a few years the per cent. of non-breeding animals is very small. Success depends on an exact early diagnosis and treatment. With the development of skill in diagnosis it is found that metritis is present in practically all cases of sterility or failure to breed and that the curing of this metritis returns the animal to the pregnant list.

The diseases of chief importance in this locality are among cattle. Many cows are saved absolutely, or preserved through treatment as breeders. The list of 32 dystocias, 60 retained placenta, 60 sterility, and 32 indigestions with so low a mortality is very suggestive. The money value of animals saved is not easy to estimate, but it is many times the expense of supporting the ambulatory clinic.

In the field of therapeutics the art of handling abnormalities of the genital system, especially metritis (abortion, retained placenta, sterility) is perhaps the most important subject in veterinary medicine at the present time.

When one considers the constant practice and skill essential to success the futility of extension work among farmers on the subject of animal diseases is obvious. Extension work in the form of individual teaching of practicing veterinarians, however, is of great importance. In human medicine, and in some countries in veterinary medicine, it is customary for men in practice to return at frequent intervals either to their own college or some other and get special information. The time required may not be more than two days to two weeks but it keeps them in touch with the most recent methods and it keeps them in training. If this custom could be established in New York State it would after a time contribute enormously to the effectiveness of the general practitioner. Men in practice obtain benefit from attending the usual lectures and recitations, probably they obtain more benefit from conferences, but individual co-operation undoubtedly contributes most to the effectiveness of the man in practice.

TABULATED REPORT OF THE AMBULATORY CLINIC JULY 1ST, 1916 TO JUNE 30, 1917

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
1. INFECTIONS AND INTOXICATIONS									
Abortion	2	*19	....	....	....	....	....	....	.....
Actinomycosis	....	3	....	....	....	....	....	2	1
Anthrax	....	1	....	....	....	....	....	1	.....
Blackleg	....	1	....	....	....	....	....	1	.....
Cowpox	....	2	....	....	2	....	....	....	.....
Dysentery, winter	....	10	....	....	10	....	....	....	.....
Glanders	1	....	....	....	....	....	....	1	.....
Hog cholera	....	....	....	14	....	....	....	14	.....
Influenza	18	....	....	....	18	....	....	....	.....
Johne's disease	....	1	....	....	....	....	....	1	.....
Keratitis, infectious	....	23	....	....	23	....	....	....	.....
Pneumonia, calf	....	12	....	....	....	....	....	6	6
Pneumonia, contagious	2	....	....	....	2	....	....	....	.....
Strangles	44	....	....	....	41	....	....	3	.....
Septicemia neonatorium	1	4	....	....	1	....	....	4	.....
Tetanus	3	....	....	1	1	....	....	3	.....
Tuberculosis	....	12	....	....	....	....	....	9	3
2. DISEASES OF THE CIRCULATORY SYSTEM									
Azoturia	7	....	....	....	5	1	....	1	.....
Heart exhaustion	5	....	....	....	1	....	3	....	1
Pericarditis, traumatic	....	4	....	....	....	....	....	4	.....
Pericarditis, tuberculous	....	1	....	....	....	....	....	1	.....
3. DISEASES OF THE RESPIRATORY SYSTEM									
Bronchitis	....	4	....	....	4	....	....	....	.....
Congestion, pulmonary	2	2	....	....	4	....	....	....	.....
Emphysema, pulmonary	....	1	....	....	....	....	....	....	1
Heaves	6	....	....	....	....	....	4	1	1
Laryngitis, catarrhal	1	6	....	....	7	....	....	....	.....
Nasal catarrh	2	2	....	....	4	....	....	....	.....
Pneumonia	1	1	....	....	2	....	....	....	.....
4. DISEASES OF THE DIGESTIVE SYSTEM									
Ascariasis	2	....	....	....	2	....	....	....	.....
Bowel catarrh, acute	4	9	....	....	13	....	....	....	.....
Choke	2	8	....	....	10	....	....	....	.....
Colic	32	3	....	....	34	....	....	1	.....
Dysphagia	....	1	....	....	1	....	....	....	.....
Enteritis	....	2	....	....	1	....	....	1	.....
Gastritis, traumatic	....	10	....	....	7	....	....	3	.....
Gastric dilatation and rupture	2	....	....	....	1	....	....	1	.....
Gastro-intestinal catarrh	10	11	....	....	21	....	....	....	.....
Impaction, colon	24	....	....	....	24	....	....	....	.....
Indigestion	14	32	....	11	54	....	....	1	2
Intussusception	....	2	....	....	2	....	....	....	.....
Meconium, retention of	1	....	....	....	1	....	....	....	.....
Paralysis, pharynx	1	1	....	....	1	....	....	1	.....
Peritonitis, perforative	2	....	....	....	....	....	....	2	.....
Prolapsus recti	....	....	....	3	2	....	....	....	1
Stenosis, intestine	1	....	....	....	....	....	....	1	.....
Stomatitis	2	....	....	....	2	....	....	....	.....
Stomach-worm disease	....	....	27	....	19	....	....	8	.....
Torsion, large colon	4	....	....	....	....	....	....	4	.....
Torsion, small intestine	1	....	....	....	....	....	....	1	.....
Tympany, acute	....	4	....	....	4	....	....	....	.....

* Cows: Conceived, 9; sold, 4; sold as non-breeders, 2; undetermined, 3; still under treatment, 1. All that were given after-treatment have conceived.

Treated and conceived 29; unknown, treated one or two times 18; treated and found incurable 2; found hopelessly sterile 4; sold, no treatment 2; sold before treatment was complete 1; died of hemorrhage after crushing the ovary 1; incomplete 3. Bull 1, mare 1; both remained sterile. It is probable that most of the unknowns conceived since they were all slightly affected.

TABULATED REPORT OF THE AMBULATORY CLINIC — *Continued*

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
5. DISEASES OF THE LIVER									
Cirrhosis, hypertrophic	1	....	....	....	1	....	....	....	.....
6. DISEASES OF THE GENITO-URINARY SYSTEM									
Cervicitis, connective tissue bands	....	1	....	....	1	....	....	....	.....
Dystocia	1	32	....	1	27	1	....	6	.....
Free martin	....	4	....	....	....	....	....	2	2
Hemorrhage, ovary	....	1	....	....	....	....	....	1	.....
Hemorrhage, prepuce	....	1	....	....	1	....	....	....	.....
Mastitis	....	22	....	3	10	1	2	2	9
Metritis, acute	....	31	....	....	21	3	....	6	1
Metritis, chronic	....	7	....	....	2	....	3	....	1
Mummified fetus	....	2	....	....	1	....	....	1	.....
Omphalitis	1	4	....	....	4	....	....	1	.....
Orchitis	1	....	....	....	1	....	....	....	.....
Paralysis, post partum	....	2	....	....	2	....	....	....	.....
Placenta, retained	....	58	....	1	57	....	....	2	.....
Pregancy, examination for	5	23	....	....	....	....	....	....	.....
Prepuce, dilatation of	....	....	....	1	....	....	....	....	.....
Prolapse, uterus	....	5	....	1	3	....	....	2	1
Prolapse, vagina	....	1	....	....	1	....	....	....	.....
Sterility	....	62	....	....	....	....	....	....	.....
Teat, stricture	....	12	....	....	8	....	....	....	4
Teat, tumor	....	1	....	....	....	....	....	....	1
Torsion, uterus	....	5	....	....	3	....	....	2	.....
Vaginitis	....	2	....	....	1	....	....	1	.....
7. DISEASES OF THE SKIN									
Dermatitis	2	5	....	....	6	....	....	....	1
Eczema	....	1	....	....	1	....	....	....	.....
Mange	4	....	....	....	4	....	....	....	.....
Pediculosis	....	7	....	....	....	....	....	....	.....
Ringworm	....	15	....	....	15	....	....	....	.....
Scratches	1	....	....	....	1	....	....	....	.....
Summer sores	1	....	....	....	1	....	....	....	.....
8. DISEASES OF THE NERVOUS SYSTEM									
Convulsions	1	....	....	....	....	....	....	1	.....
Dummies	1	....	....	....	....	....	....	1	.....
Encephalitis	....	1	....	....	....	....	....	1	.....
Heat stroke	2	....	....	....	2	....	....	....	.....
Paraplegia	2	....	....	1	....	....	....	1	2
Parturient paresis	....	10	....	....	10	....	....	....	.....
Thumps, abdominal	1	....	....	....	1	....	....	....	.....
Tremors	....	1	....	....	1	....	....	....	.....
9. POISONING									
Arsenate of lead	....	4	....	....	....	....	....	4	.....
Bittersweet	....	1	....	....	1	....	....	....	.....
Butternut twigs	....	3	....	....	3	....	....	....	.....
Mouldy oats, chaff, etc	3	1	....	....	1	....	....	3	.....
10. DISEASES OF THE EYE									
Keratitis, infectious	....	6	....	....	6	....	....	....	.....
Periodic ophthalmia	10	....	....	....	2	1	3	....	.....
Wounds	3	....	....	....	1	....	....	....	.....
11. EXTERNAL DISEASES									
Abscess	11	15	....	....	26	....	....	....	.....
Arthritis	4	....	....	....	1	1	1	1	.....
Carpitis	1	....	....	....	....	....	....	....	1
Castration	19	14	10	88	87	....	....	1	.....
Contusions	4	....	....	....	4	....	....	....	.....
Contracted hoof	1	....	....	....	....	....	....	....	1
Fistula	4	1	....	....	4	....	....	1	.....
Footrot	....	12	....	....	12	....	....	....	.....

TABULATED REPORT OF THE AMBULATORY CLINIC — *Concluded*

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
Fracture, carpus	....	1	....	....	....	....	....	1	.....
Fracture, humerus	1	....	....	....	....	....	....	1	.....
Fracture, ilium, ext. ang	1	....	....	....	1	....	....	....	.....
Fracture, pelvis	1	....	....	....	1	....	....	....	.....
Fracture, phalanx, first	1	....	....	....	....	....	....	1	.....
Fracture, radius	2	....	....	....	....	....	....	2	.....
Fracture, tibia	....	1	....	....	1	....	....	....	.....
Fracture, ulna	1	....	....	....	1	....	....	....	.....
Gingivitis	....	1	....	....	1	....	....	....	.....
Gall, shoulder	1	....	....	....	1	....	....	....	.....
Gonitis	2	6	....	....	1	....	4	2	1
Hematoma	4	....	....	....	4	....	....	....	.....
Hernia, inguinal	....	....	....	7	....	....	....	....	.....
Hernia, umbilical	2	....	....	....	7	....	....	....	.....
Hernia, ventral	....	2	....	....	....	....	2	....	.....
Lameness, shoulder	2	....	....	....	2	....	....	....	.....
Laminitis	1	....	....	....	....	....	....	1	.....
Lymphangitis	7	....	....	....	7	....	....	....	.....
Navicular disease	2	....	....	....	....	....	....	....	2
Open joint, digital	1	....	....	....	....	....	....	1	.....
Paralysis, suprascapular	2	....	....	....	2	....	....	....	.....
Paralysis, radial	1	....	....	....	....	....	....	1	.....
Pododermatitis	7	2	....	....	9	....	....	....	.....
Pododermatitis, suppurative corn	3	....	....	....	3	....	....	....	.....
Paraplegia	....	4	....	....	2	....	....	2	.....
Quittor	1	....	....	....	1	....	....	....	.....
Ringbone	1	....	....	....	....	....	....	....	1
Spavin	1	....	....	....	....	1	....	....	.....
Sprain, fetlock	1	....	....	....	....	....	....	....	1
Strain, tendon	4	....	....	....	4	....	....	....	.....
Stiffness, general	1	1	....	....	2	....	....	....	.....
Teeth, long	4	....	....	....	....	....	....	....	.....
Teeth, sharp	30	....	....	....	....	....	....	....	.....
Teeth, split	6	....	....	....	....	....	....	....	.....
Teeth, extracted	1	....	....	....	....	....	....	....	.....
Tumors	2	2	....	....	4	....	....	....	.....
Thrush	3	....	....	....	3	....	....	....	.....
Wounds	2	1	....	....	3	....	....	....	.....
Wounds, castration	3	....	....	....	3	....	....	....	.....
Wounds, foot	7	1	....	....	8	....	....	....	.....
Wounds, head and body	3	....	....	....	3	....	....	....	.....
Wounds, limbs	21	1	....	....	18	2	....	1	.....
Wounds, nail	10	....	....	....	10	....	....	....	.....
Wounds, nailing	2	....	....	....	2	....	....	....	.....
Wounds, teat	....	8	....	....	8	....	....	....	.....
12. MISCELLANEOUS									
Anthrax, immunization	....	28	....	....	....	....	....	....	.....
Amputation of tails	....	....	20	....	....	....	....	....	.....
Cast in stable	7	4	....	....	10	....	....	1	.....
Dehorning	....	163	....	....	....	....	....	....	.....
Exhaustion	3	1	....	....	3	....	....	1	.....
Hog-cholera, immunization	....	....	....	1,204	....	....	....	....	.....
Tuberculin test	....	60	....	....	....	....	....	....	.....
Ringing bulls	....	2	....	....	....	....	....	....	.....
Soundness examination	9	2	....	....	....	....	....	....	.....
Sputum examination	....	3	....	....	....	....	....	....	.....
Total	445	859	57	1,336	....	....	....	....	.....
Gross total	....	....	....	....	....	....	....	2,694	.....
Counted twice	....	....	....	....	....	....	....	42	.....
Net total	....	....	....	....	....	....	....	2,652	.....

REPORT OF THE SURGICAL AND CONSULTING CLINIC FROM JULY 1, 1916, TO JUNE 30, 1917

J. N. FROST and F. C. CLARK

Department of Surgery

The surgical and consulting clinics have been up to their previous standards both as to variety and number of cases treated. Anesthetics have been used in all except the very minor cases. By the proper administration of local anesthetics they have replaced to a large extent the use of general anesthesia. Where general anesthesia was necessary, the anoci-association method was used.

The large number of cases presented for the treatment of ophthalmia or what is known as moon blindness has afforded an opportunity to try new methods of treatment in an endeavor to increase the percentage of recoveries.

The increasing importance of dairy animals has led to the use of what little time and money could be spared for experimental work in abdominal surgery and disease of the udder. As a result we have been able to successfully remove portions of the small intestine in cases of intussusception in cattle and to provide a treatment for mastitis which has given satisfactory results.

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
Abscess	20	4	1	1	23	....	....	2	1
Actinomycosis	....	2	....	....	1	....	....	1	.....
Alveolus — Tumor of	3	....	....	....	....	....	....	3	.....
Angioma — Nasal cavity	1	....	....	....	....	....	....	1	.....
Anus, fistula of	1	....	....	....	....	....	1	....	.....
Arthritis, acute	1	1	....	....	2	....	....	....	.....
Arthritis, purulent	2	1	....	....	2	....	....	1	.....
Arthritis, chronic	30	1	....	....	20	2	1	....	8
Azoturia	4	....	....	....	4	....	....	....	.....
Bronchitis	3	....	....	....	3	....	....	....	.....
Canker	1	....	....	....	....	....	1	....	.....
Carpitis	1	....	....	....	....	....	1	....	.....
Castrations	23	2	....	35	60	....	....	....	.....
Cataract	2	....	....	....	....	....	2	....	.....
Caudal myectomy	1	....	....	....	1	....	....	....	.....
Choke	1	....	....	....	1	....	....	....	.....
Colic	3	....	....	....	3	....	....	....	.....
Colon, torsion of	1	....	....	....	....	....	....	1	.....
Contracted foot	1	....	....	....	....	1	....	....	.....
Contusion	5	1	....	....	6	....	....	....	.....
Corn	6	....	....	....	6	....	....	....	.....
Cornea, wound of	2	....	....	....	2	....	....	....	.....
Cryptorchid	6	....	....	1	7	....	....	....	.....
Curb	3	....	....	....	3	....	....	....	.....
Cystitis, purulent	....	1	....	....	1	....	....	....	.....
Dehorning	....	1	....	....	1	....	....	....	.....
Dermatitis	5	....	....	....	5	....	....	....	.....
Diarrhea	5	3	....	....	8	....	....	....	.....
Dysuria	1	....	....	....	1	....	....	....	.....
Eczema	5	....	....	....	5	....	....	....	.....
Edema	1	....	....	....	1	....	....	....	.....
Empyema, sinus	5	3	....	....	8	....	....	....	.....
Entropium	2	....	....	....	2	....	....	....	.....
Examination for pregnancy	....	3	....	....	....	....	....	....	.....
Examination for soundness	7	....	....	....	....	....	....	....	.....
Eye, prolapse of	....	....	1	....	1	....	....	....	.....
Eye, removed	....	1	....	....	1	....	....	....	.....
Fibroma	2	....	....	....	2	....	....	....	.....
Fistula anus	1	....	....	....	....	....	1	....	.....
Fistula teat	....	1	....	....	1	....	....	....	.....
Fistulous withers	18	....	....	....	13	....	....	1	4
Flexor pedis, rupture of	1	....	....	....	....	....	....	1	.....
Follicular cyst	2	....	....	....	2	....	....	....	.....
Foot contracted	1	....	....	....	....	1	....	....	.....
Foot deformed	1	....	....	....	....	....	1	....	.....
Fracture, rud., metacarp	1	....	....	....	1	....	....	....	.....
Fracture, nasal bone	1	....	....	....	1	....	....	....	.....
Glossitis	1	....	....	....	1	....	....	....	.....
Gonitis	5	1	....	....	....	2	4	....	.....
Heaves	4	....	....	....	....	4	....	....	.....
Hematome	6	....	....	....	6	....	....	....	.....
Hemorrhage, nostril	1	....	....	....	1	....	....	....	.....
Hernia, scrotal	....	....	....	29	29	....	....	....	.....
Hernia, strangulated	....	....	....	2	2	....	....	....	.....
Hernia, umbilicus	....	3	....	2	3	....	2	....	.....
Hernia, ventral	2	....	....	....	....	....	2	....	.....
Horn tumor	2	....	....	....	2	....	....	....	.....
Horse pox	1	....	....	....	1	....	....	....	.....
Impaction	1	....	....	....	1	....	....	....	.....
Indigestion, acute	2	....	....	....	2	....	....	....	.....
Intussusception	....	3	....	....	3	....	....	....	.....
Iritis	3	....	....	....	3	....	....	....	.....
Johne's disease	....	1	....	....	....	....	....	1	.....
Joint opened	2	1	....	....	2	....	....	1	.....
Laminitis	4	....	....	....	4	....	....	....	.....
Lymphangitis	2	....	....	....	2	....	....	....	.....
Malignant tumor	10	....	....	....	2	....	....	8	.....
Mange	2	....	....	....	2	....	....	....	.....
Mastitis	....	4	....	....	4	....	....	....	.....

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
Melanoma	2	....	....	....	1	....	....	1	.....
Metritis	9	5	....	....	14	....	....	....	.....
Myectomy, caudal	1	....	....	....	1	....	....	....	.....
Nasal bone, fracture of	1	....	....	....	1	....	....	....	.....
Navicular disease	12	....	....	....	....	12	....	....	.....
Odontome	2	....	....	....	2	....	....	....	.....
Open joint	2	1	....	....	2	....	....	1	.....
Open tendon sheath	1	....	....	....	1	....	....	....	.....
Ophthalmia	1	....	....	....	....	1	....	....	56
Os pedis, sinking of	91	....	....	....	30	....	5	....	.....
Papilloma	3	....	....	....	3	....	....	....	.....
Paralysis, pharynx	1	....	....	....	....	....	....	1	.....
Parasites	16	....	9	....	23	....	....	2	.....
Penis, tumor of	1	....	....	....	1	....	....	....	.....
Pharyngitis	1	....	....	....	1	....	....	....	.....
Pharynx, paralysis of	1	....	....	....	....	....	....	1	.....
Phymosis	1	1	....	....	2	....	....	....	.....
Pink eye	3	....	....	....	3	....	....	....	.....
Placenta retained	1	2	....	....	3	....	....	....	.....
Pododermatitis	4	2	....	....	6	....	....	....	.....
Poll evil	13	....	....	....	13	....	....	....	.....
Pregnancy, examination for	....	3	....	....	....	....	....	....	.....
Prolapse, eye	....	....	1	....	1	....	....	....	.....
Prolapse, rectum	....	....	....	1	1	....	....	....	.....
Prolapse, uterus	....	....	....	1	1	....	....	....	.....
Pyemia	1	1	....	....	....	....	....	2	.....
Pyometra	5	5	....	....	10	....	....	....	.....
Quarter crack	2	....	....	....	2	....	....	....	.....
Quittor	6	....	....	....	6	....	....	....	.....
Rectum, prolapse of	....	....	....	1	1	....	....	....	.....
Retained placenta	1	2	....	....	3	....	....	....	.....
Resection, flexor pedis	1	....	....	....	1	....	....	....	.....
Ringbone	10	....	....	....	5	....	1	....	4
Ringworm	1	1	....	....	2	....	....	....	.....
Roaring	9	....	....	....	7	....	....	....	2
Rupture, ext. pedis	1	....	....	....	....	....	....	1	.....
Scratches	2	....	....	....	2	....	....	....	.....
Scrotal hernia	....	....	....	29	29	....	....	....	.....
Side bones	3	....	....	....	....	3	....	....	.....
Spavin	24	....	....	....	10	....	1	....	13
Spaying	6	....	....	....	6	....	....	....	.....
Splint	4	....	....	....	4	....	....	....	.....
Sprain, biceps	1	....	....	....	1	....	....	....	.....
Sprain, check ligments	3	....	....	....	2	1	....	....	.....
Sterility	7	10	....	....	11	....	....	1	5
Strangles, abscess	4	....	....	....	4	....	....	....	.....
Strangulated hernia	....	....	....	2	2	....	....	....	.....
Sweeney	2	....	....	....	2	....	....	....	.....
Synovitis, purulent	2	1	....	....	2	....	....	1	.....
Teat, contusion of	....	1	....	....	1	....	....	....	.....
Teat, fistula of	....	1	....	....	1	....	....	....	.....
Teeth, examination of	88	....	....	....	....	....	....	....	.....
Teeth, wolf	5	....	....	....	....	....	....	....	.....
Teeth, diseased	18	1	....	....	19	....	....	....	.....
Teeth, long	6	....	....	....	6	....	....	....	.....
Tendon, sheath open	1	....	....	....	1	....	....	....	.....
Tetanus	1	....	....	....	....	....	....	1	.....
Thoroughpin	2	....	....	....	2	....	....	....	.....
Thrush	2	....	....	....	2	....	....	....	.....
Torsion, colon	1	....	....	....	....	....	....	1	.....
Torsion, uterus	....	1	....	....	....	....	....	1	.....
Trochanteric lameness	2	....	....	....	2	....	....	....	.....
Tuberculosis	....	2	....	....	....	....	....	2	.....
Tumor, benign	25	1	....	....	26	....	....	....	.....
Tumor, malignant	10	....	....	....	2	....	....	8	.....
Umbilicus, abscess of	1	....	....	1	1	....	....	1	.....

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
Umbilicus, hernia of	....	3	....	2	3	....	2	....	.....
Uterus, prolapse of	....	....	....	1	1	....	....	....	.....
Uterus, torsion of	....	1	....	....	....	....	....	1	.....
Withers, abscess of	2	....	....	....	2	....	....	....	.....
Withers, fistula of	18	....	....	....	13	....	....	1	4
Withers, hematome of	1	....	....	....	1	....	....	....	.....
Wounds	43	2	....	....	45	....	....	....	.....
Post mortems	10	7	2	....	....	....	....	....	.....
Total	726	92	14	108	618	27	25	48	97
Gross total									940
Counted twice and post mortems									180
Net total									760

REPORT OF THE CLINIC FOR SMALL ANIMALS FOR 1916–1917

H. J. MILKS and W. E. MULDOON

Department of Materia Medica and Small Animal Clinic

There has been the usual number of cases in this clinic during the past year. The material has been very valuable for purposes of instruction so that it has been possible to round out a number of subjects. It is well known that only general principles can be pointed out in class room work in the subjects of medicine, surgery and therapeutics and that these subjects can be best studied and demonstrated in clinics. Students have been drilled in diagnosis, microscopic examination of feces and skin for parasites and in the theory and actual practice of the treatment of sick and injured animals. Furthermore, they are required to keep notes on each case, including the symptons, diagnosis and treatment. Since most of the animals remain in the clinic for some time it is possible to follow the entire course of the disease.

EXTERNAL DISEASES

Diseases of the Eye

	Dog	Cat	Total
Atrophy of eye	1	.....	
Conjunctivitis	2	.....	
Cornea, opacity of	5	.....	
Entropion	1	.....	
Eye ball, enucleation of	1	.....	
Keratitis	6	.....	
Orbital gland, removal of	3	.....	
Wound of lid (rabbit)	....	1	
	19	1	
			20

Head and Neck

	Dog	Cat	Total
Abscess of jaw	....	1	
Abscess of neck	....	1	
Carcinoma of ear	1	.....	
Carcinoma of thyroid	1	.....	
Deafness	1	.....	
Fractured jaw	3	.....	
Foreign body in mouth	1	.....	
Glossitis	1	.....	
Tumor of tongue	1	.....	
Teeth, clipping of (ferret)	1	.....	
Tooth fistula	....	1	
Tumefied submaxillary gland	1	.....	
Otorrhea, catarrhal	3	.....	
Otorrhea, parasitic	9	2	
Oestrus in head (rabbit)	....	1	
Oestrus in neck	....	1	
Wound on ear	3	.....	
Wound on jaw	2	1	
Wound on neck	2	2	
	30	10	
			40

Anterior Extremities

	Dog	Cat	Total
Amputation of foot	....	1	
Abscess of shoulder	1	.....	
Atrophy of shoulder	1	.....	
Radius and ulna, fracture of	1	.....	
Injury to leg	2	.....	
Radial paralysis	1	.....	
Wound on shoulder	2	.....	
Wound on leg	1	2	
Wound on foot	2	2	
	11	5	
			16

Thorax and Abdomen

	Dog	Cat	Total
Castration	5	13	
Carcinoma of mammary gland	1	.....	
Hernia, umbilical	4	1	
Oophorectomy	115	36	
Abscess of anal pouches	6	.....	
Abscess of inguinal lymph gland	1	.....	
Ascites	1	.....	
Carcinoma of inguinal lymph gland	1	.....	
Caesarian section	1	.....	
Abscess on side	1	.....	
Wound on side	3	.....	
Proctitis	1	.....	
	140	50	
			190

Posterior Extremities

	Dog	Cat	Total
Amputation of toe (fowl)	....	1	
Amputation of leg	....	1	
Luxation of patella	1	.....	
Fracture of femur	4	1	
Fracture of tibia and fibula	1	.....	
Paralysis	6	.....	
Nails, ingrowing	1	.....	
Wound, hip	3	1	
Wound, hock	4	0	
Wound, foot	2	.....	
	22	4	
			26

Vertebrae, Pelvis and Tail

	Dog	Cat	Total
Amputation of tail	10	0	
Wound of tail	3	1	
	13	1	
			14

Miscellaneous

	Dog	Cat	Total
Autopsy	....	1	1

Internal Diseases

Infectious Diseases

	Dog	Cat	Total
Distemper	35	.....	
Infectious enteritis	....	2	
Hemorrhagic gastroenteritis	3	.....	
Tuberculosis	....	1	
	38	3	
			41

Circulating System

	Dog	Cat	Total
Heart disease	2	.....	2

Respiratory System

	Dog	Cat	Total
Catarrh, nasal	1	1	
Laryngitis	1	.....	
Bronchitis	....	1	
Pneumonia	....	1	
	2	3	
			5

Digestive System

	Dog	Cat	Total
Gastritis	10	2	
Impaction of crop (fowl)	....	1	
Intestinal catarrh	2	1	
Indigestion	3	1	
Constipation	2	.....	
Worms, hook (uncinaria)	7	.....	
Worms, round	7	2	
Worms, tape	2	.....	
	33	7	
			40

Genito-Urinary System

	Dog	Cat	Total
Abscess of prostate	1	.....	
Ruptured kidney	1	.....	
Infectious genital tumors	2	.....	
Incontinence of urine	2	.....	
Urethral calculus	2	.....	
Prepuce, ulcer of	1	.....	
Vagina, tumor of	1	.....	
Vagina, prolapse of	1	.....	
Vaginitis	1	.....	
Metritis	1	.....	
	13		13

Diseases of the Skin

	Dog	Cat	Total
Urticaria	1	.....	
Mange, sarcoptic	3	6	
Mange, follicular	10	.....	
Eczema	15	1	
Fleas	21	.....	
Lice	2	.....	
Shedding hair	1	.....	
Papilloma	1	.....	
	54	7	
			61

Nervous System

	Dog	Cat	Total
Chorea	1	.....	
Epilepsy	3	.....	
	4		
			4

Miscellaneous

	Dog	Cat	Total
Rachitis	3	.....	
Suspected rabies	1	.....	
Destroy	3	5	
Observation	9	.....	
	16	5	
			21

Summary

	Dog	Cat	Ferret	Rabbit	Foal	Total
Diseases of the eye	19	....	....	1	....	20
Head and neck	29	9	1	1	....	40
Anterior extremities	11	5	....	....	....	16
Thorax and abdomen	140	50	....	....	....	190
Posterior extremities	22	3	....	....	1	26
Vertebrae, pelvis and tail	13	1	....	....	....	14
Miscellaneous	..	1	....	....	....	1
Infectious	38	3	....	....	....	41
Circulatory system	2	....	....	....	....	2
Respiratory system	2	3	....	....	....	5
Digestive system	33	6	....	....	1	40
Genito-urinary system	13	....	....	....	....	13
Diseases of the skin	54	7	....	....	....	61
Nervous system	4	....	....	....	....	4
Miscellaneous	16	5	....	....	....	21
	396	93	1	2	2	494

Total number of cases	494
Counted twice	10
Number of animals	484

RESEARCHES IN THE DISEASES OF BREEDING CATTLE [1]

By W. L. WILLIAMS and W. A. HAGAN [2]

Department of Obstretrics and of Research upon the Diseases of Breeding Cattle

In prior annual reports I have attempted to outline the scope and significance of the term "contagious abortion" as understood by me. It has been endeavored constantly to overcome the common conception that there is, or can be, in cattle or other mammals, an infection or disease which can do harm only by causing abortion and which, when affecting an animal, necessarily or generally causes abortion. It has been aimed also to avoid the unique and unstable position that an infectious disease can or does exist which may affect the female alone, or conjointly the fetus in utero, and only in the presence of pregnancy. Pregnancy may and does complicate and intensify uterine and other female genital diseases, especially those due to infection. When there exists an infection in the cervix or in the uterus prior to conception, it may cause sterility, or if conception occurs the fetal envelopes, and later the fetus itself, tend to become involved by contiguity. The infection then usually follows a split course. When the fetus is expelled, alive or dead, mature or immature, some of the infection tends to accompany the fetus (intra-uterine infection of the young) and some to persist in the uterus or other portions of the genital tract.

It has been held, in all reports by this department, that "contagious abortion" in cattle (and in other animals) is a chronic infection, invading chiefly the genital tract of each sex and causing lesions which may result in sterility, abortion, premature birth, and retained afterbirth. Recently it has proven necessary definitely to include in this group certain highly destructive diseases of calves. Taken singly, each of the disasters cited above

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[1] The Bureau of Animal Industry, U. S. Department of Agriculture, cooperating.

[2] The body of this report and the section upon *tuberculosis of the genital organs of cattle* have been prepared by W. L. Williams. The section upon the *etiology and mode of infection of white scours*, and the studies upon which it is based, are the special work of W. A. Hagan. In general, the research work has been conducted jointly.

causes very considerable losses; taken together as the result of one basic infection or group of infections, it assumes a far greater importance to cattle breeders and to the State.

The infection which is regarded as causing "contagious abortion" is presumed to play a highly important part in the diseases of the ovaries — chiefly cystic degeneration of the corpus luteum or yellow body, and cystic disease of the ovisacs, or Graafian follicles. No connection between the infection of contagious abortion and the cystic changes in the ovaries has been clearly demonstrated technically, but there are abundant clinical facts to warrant the assumption of a vital relation between ovarian sterility and "contagious abortion," until otherwise determined.

In the oviducts, uterus, cervix, and vagina, infection with inflammation is common. When the infection is confined to the oviducts, it tends to cause sterility by blocking the canals so that the spermatozoa of the male can not pass up to the ovary to meet and fertilize the egg, or ovum, at the time of escape from the ovary. If the egg should become fertilized, it is still liable to be destroyed by the infection while passing downwards to the cavity of the uterus. If the uterine cavity contains infection and the mucous membrane is inflamed, the fertilized egg, if it reaches the uterine cavity, may be promptly killed. The resultant failure to reproduce is called sterility.

The inflammation resulting from the infection does not generally begin, or have its only habitat, in the central part of the uterine cavity. The uterus of the cow consists of a small *body* and two *horns*. Each horn is sharply conical; the apex is continuous with the oviduct and the base with the uterine body. The cavity of the body of the uterus ends posteriorly in the constricted cervical canal. The narrow apex of each horn and the narrow, intricate canal of the cervix offer favorable points in which infection may lodge and from which it may spread. When located at the apex of either horn, the infection tends to cause sterility, but can not so readily induce abortion.

Abortion — the expulsion from the uterus of an immature dead fetus — is always caused, so far as known, when dependent upon infection, by an irritation or inflammation at the *vaginal* end of the uterus, which causes a contraction of the comparatively healthy

walls of the anterior, or ovarian end. When the disease is located at the anterior, or ovarian end of the uterus, the walls at that point become paralyzed and powerless to push the fetus toward and into the vagina. There has been entirely too much mystery thrown about the phenomenon of abortion. In every case recorded where a cow (or other animal) has been destroyed promptly after she has aborted or given birth prematurely, there has been observed an inflammation (metritis and cervicitis) evidently due to infection, which had its chief centre at the os uteri internum, where the canal of the cervix opens into the uterine cavity. From that point, the inflammation has radiated forward toward the ovarian end of the uterine cavity. Thus abortion is induced. Too often it has been assumed that abortion occurs otherwise.

It has been alleged that certain drugs (ecbolics) given a pregnant animal may induce uterine contractions, in the absence of intrauterine infection, and cause abortion, but the allegation has not been proven, and no reliable evidence has been submitted that any drug can alone cause the premature expulsion of the fetus.

Apparently it has been thought also that bacteria or their toxins entering the blood in sufficient volume may cause contraction of the uterine walls, with death and expulsion of the fetus, without the abortion bacilli having entered the uterine cavity and established an inflammation at its vaginal end. All autopsies directly deny this. It is the infection within the cervical canal and uterine cavity — the cervicitis and metritis — not the abortion, which fundamentally interests us. The intra-uterine and cervical infection — the metritis and cervicitis — do not belong alone to pregnancy, but may and do exist at any and every period of female life. Accordingly we have for a long time insisted that the infection may and does exist frequently in the cervical canal of a heifer which has not been bred, and causes sterility. In the report of the college for 1915–1916, it has been shown that infection invades the organs of the fetus from the diseased uterus of its dam. This is shown still more definitely in Dr. Hagan's work, "The Etiology and Mode of Infection in White Scours of Calves," constituting a section of this report. In the report of 1914–1915, evidence is submitted to show that many calves acquire the infection with the Bang abortion organism when fifteen to twenty days old,

through contaminated milk, and further that the infection may be traced through the first two years of the heifer's life, when it may finally cause sterility, abortion, retained placenta, or other disaster resulting from the presence of the infection within the uterine cavity. If a calf is born, the infection, which has invaded the fetus from the diseased uterus of the mother, may cause in the new-born calf septicaemia, calf scours, or pneumonia, thus establishing an endless chain of infection, generation after generation.

Dairying and intensified breeding have served to extend and increase the virulence of the infection. This department insists that the spread of "contagious abortion" is accomplished principally through two agencies — infection of the fetus from the diseased uterus or of the new-born calf by contaminated milk, and infection by sexual contact between animals having diseased genital organs.

We have insisted constantly that disinfectants given internally to pregnant animals or applied to their posterior parts, or to the gutters, floors, or other parts of the stable, can not disinfect the interior of the uterus or affect favorably the sterility-abortion-white scours group of diseases. Instead, we have insisted that in any effort at control the genital system of the cow must be made as clean as possible, prior to service by an equally clean bull, in order that the cow shall breed promptly, her pregnancy continue naturally to full term, and finally a healthy vigorous calf be born. Once the calf has been born healthy, its health should be scrupulously guarded by correct feeding and handling.

There is no important disease of domestic animals regarding the nature of which there are so many conflicting opinions. In 1911–1912 I advocated the above views, insisting that the infection which often leads to abortion is virtually universal. This view was then almost universally rejected by veterinarians, cattle breeders, and dairymen. Observations and researches have gradually compelled thoughtful men either to accept or to consider seriously the views then announced. In a large measure, the modification in view has been brought about by the breaking down of all measures for control based upon other hypotheses. Most persons have held that the infection is not at all universal. They

admit that some or many herds have the infection, but state that this, that or the other herd is free. The owner believes implicitly that *his* herd is free and that all he needs do is to avoid introducing from elsewhere an *aborter*. While he is searching the horizon for approaching danger from without, the infection within his own herd stealthily acquires intensity and virulence, until finally the storm breaks in his own stable. Then he searches diligently for a scapegoat. Possibly he can recall the introduction of a cow from an aborting herd, but she does not always fill the office satisfactorily. In the long run, the breeder or dairyman is forced to one of two conclusions: either the infection is virtually universal, not always causing visible harm; or there is no secure method of keeping the infection out of a sound herd. Practically, the two propositions reach the same goal. The breeder must exert eternal vigilance to prevent losses. The general belief that only some herds are infected has led to serious disaster. It is responsible for the attempt to control the infection by the temporary isolation of aborters. Only part of the aborters are recognized. Almost all abortions before the fifth month of pregnancy are unseen, and most of those during the ninth month are not recognized. Probably not more than fifty per cent. of actual abortions are seen, though evidently the unrecognized aborter is as dangerous as the recognized one. The aborters which are recognized always give off abundant infection before aborting, probably for weeks or months, and consequently prior to isolation. If isolated, there is no reliable way by which it can be determined when, if ever, the aborter becomes free from the infection and ready to be returned to the stable.

Only a small proportion of the cattle having abortion infection in the uterine cavity are observed to abort. More of them suffer from sterility, calve prematurely, have retained afterbirth, or have a discharge from the uterus following calving. The infection, so far as known, is identical in all cases, but the disaster of abortion occurs only in one. The other types of disease are equally significant.

The isolation of aborters has long been regarded by many as the most reliable agency in the control of the losses. Throughout, I have opposed this view and held that isolation must prove ineffi-

cient and generally harmful, because in contagious abortion it fails to meet three basic requirements in the control of contagious diseases by isolation:

(1) The isolation shall include all individuals which are giving off the contagion.

(2) The isolation must occur prior to the discharge of the contagion by the patient.

(3) The isolation must continue to the end of the period when the individual can spread the contagion.

The isolation of aborters fulfills none of these conditions and makes no approach thereto. On the other hand, the isolation of aborters is beset with two great dangers:

(1) Breeders trust erroneously to isolation as an effective measure of control and neglect highly essential measures.

(2) An isolated aborter is a sadly neglected animal. I have seen many aborters in quarantine, and have yet to see the first one placed in as good quarters as those from which she was removed. According to the usual view that the caretaker of such an animal must change his clothing, especially his shoes, and disinfect his hands before coming in contact with the other animals of the herd (which have the same infection) the poorest caretaker about the establishment is detailed to care for the unfortunate individual. She gets poor care, and finally returns to the stable as dangerous as when she left it.

A diseased animal, whatever the nature of the malady, should either be destroyed as a menace to other animals or given especially good housing and care in order to restore her to health as certainly and promptly as possible. I have never observed this plan with isolated aborters.

I have been in conflict also with the common view that the abortion infection produces immunity. Some say that an animal which has aborted one year is not as liable to abort the next. This is a confusing statement. If a certain cow aborts one year, that is a positive occurrence. She can be neither more nor less liable to abort the following year. Two facts stand out clearly. In most herds there is a greater tendency to abortion in first than in subsequent pregnancies. In a few herds the liability is greatest in second pregnancy. After the first pregnancy has terminated, sta-

tistics show most emphatically and uniformly that those animals in a herd which have aborted during a given year are more liable to abort the next year than those in the same herd which calved physiologically (without premature birth, retained afterbirth, etc.) during the prior year. Admittedly, many animals abort one year and do not abort the next. Otherwise, the problem of controlling the disease would be hopeless. Many animals which abort die from complications, and a far greater number become hopelessly sterile. On the whole, probably twenty to thirty per cent. of aborters do not have an opportunity to abort a second time. If an animal aborts twice, she becomes too unprofitable, except in highly pedigreed stock, to warrant retention in the herd. That is one reason why few cows abort more than twice. I have record of a cow which either aborted, calved prematurely, or was sterile for five successive years, and died from her disaster in her fifth breeding year. All her life she was inefficient, and from a dairying or breeding standpoint a hopeless bankrupt. Could her career have been foreseen, of course she would not have been permitted to live beyond veal age.

The view that immunity is acquired through aborting has led to much conflict of opinion in the control of the disease. I have held that contagious abortion is a chronic infection and that consequently there can be no immunity comparable to that observed in many acute infectious diseases. I have held that the outstanding distinction between acute and chronic infections lies in the fact that in an acute disease either the patient or the disease-producing organism must perish, while in a chronic infection both the patient and the invader may live indefinitely. If the patient survives an acute infection, not only does the invader die, but the patient acquires the power to resist more or less definitely a second invasion by the same infection, as, for example, smallpox. This power is known as immunity. In a chronic infection, the invader may continue indefinitely in the system of the patient. Under stress (pregnancy, bad food, housing, labor, etc.) the invader may acquire increased intensity and destroy the life of the patient. Under favorable environment, the patient may acquire distinct ascendancy over the infection so that, while the invader may continue to exist, the patient, though still infected,

recovers efficiency. The infection may become isolated (encapsuled tubercle) or may become so suppressed that its presence is no longer recognizable, but may be re-invigorated (dormant syphilis rendered active by venery) or the system may acquire complete mastery over the infection and regain soundness, but under unfavorable conditions reinfection may follow.

Because I have regarded contagious abortion as a distinctively chronic infection, incapable of inducing a true immunity, I have come into conflict with many in reference to the control of the disease. Various methods of immunization have been industriously championed by many, especially those financially interested in alleged immunizing sera or bacterins. The conflict has led to two diametrically opposite plans of handling. I have advocated the raising of the animal's resistance by reducing to the available minimum the volume and virulence of the infection to which the patient is exposed and increasing to the maximum the power of resistance by judicious feeding, housing, exercise, and other hygienic elements conducive to health. I have advocated two definite correlated measures of control:

(1) Prior to permitting the female to be served by the male, her genital tract is to be critically examined, and disinfected if necessary (mechanically or chemically) as completely as practicable, in order that she shall not be sterile, but shall conceive promptly; that the embryo shall not perish (abortion); that the infection in the uterus shall not cause premature birth, retained afterbirth, or metritis; and that the calf shall not be born already infected with "contagious abortion" (calf septicaemia, calf scours, calf pneumonia, chronic indigestion, and unthrifty condition).

(2) After the calf has been born, it should be fed judiciously upon healthful milk and kept apart from other calves which are, or may be diseased. If the calf becomes diseased, it should be vigorously handled and promptly restored to health. Thus I advocate the minimizing of infection and the invigoration thereby of the patient, as opposed to the intensification of the infection, or intoxication, advocated by many.

Those who believe that abortion induces immunity have attempted to establish artifically such immunity in a manner to

circumvent abortion. They have tried to induce immunity in pregnant animals with killed bacteria (bacterins) and to immunize the non-pregnant cow by hyper-infection prior to conception. We have tried a third plan, purely as an experiment, which was unsuccessful, the temporary immunization with a serum. Like the isolation of aborters, immunization has proven a pitfall and caused its advocates to postpone or exclude fundamental hygienic principles. If a bit of liquid squirted under the skin will protect against abortion, why resort to the labor and care necessary to the hygienic handling of cattle?

Regarding the act of abortion as the disease has led many to have faith in drugs. More than fifty years ago, carbolic acid was recommended as a cure, and has been spasmodically recommended by individuals ever since, but it has naturally failed in every critical test. Later, methylene blue was highly recommended, and has as promptly failed.

The failure of the isolation of aborters, of vaccines, bacterins, and sera, and of drugs given internally to control the disease, and the continued advocacy of each by some persons have served to render the problem of the control of contagious abortion one of the most perplexing and discouraging tasks the cattle breeder and dairyman must face. In the meantime, the losses from the diseases are growing annually. The large dairies are frequently conducted at a monetary loss. The chief element in causing the deficit is the sterility-abortion-calf scours group of diseases. The breeding herds of pedigreed dairy cattle generally suffer extremely from this group of diseases. In these it is rare to find more than sixty living young under one year of age per one hundred females of breeding age. It is by no means rare to find herds in which the ratio of calves under one year to females of breeding age falls below thirty per cent. A dairy herd must have a high breeding efficiency, or it can not be made profitable. When the ratio of abortion, sterility, and calf scours, singly or collectively, is high, the dairying efficiency is low. When this group of diseases is virulent, cows which calve apparently normally fail to reach that dairying efficiency which makes success. Perhaps the afterbirth is retained or there is a more or less marked uterine discharge and the cow fails to produce milk of satisfactory quantity or quality.

The outbreak of the world war has served to accentuate the unfavorable conditions of dairying and cattle breeding. The increased cost of cattle foods and of labor has precipitated a crisis in a highly important industry, which has long been unprofitable and unsatisfactory to a large proportion of those engaged in it. Frantic appeals are now being made for the conservation of dairy cows. The dairymen are urged to hold their cows, as a patriotic duty, even at a financial sacrifice. They are encouraged to believe that after the war, at the latest, conditions will be so adjusted that dairying will be profitable. The fact that dairying, prior to the war, was not generally profitable is apparently ignored, and no reason is given why the industry, so largely unprofitable because of diseased animals, will be more profitable after than before the war. Dairymen are not generally sacrificing healthy cows which are yielding an abundance of good milk. Very largely, they are being forced to sell unprofitable cows, which earlier, with the lower cost of food stuffs, they might have dared keep in hope that ultimately they would prove profitable. At war prices, a dry cow not due to calve for several months is too great a burden to keep. If kept, the very fact that she failed to conceive promptly, that she was temporarily sterile, renders it probable that she will abort or have retained afterbirth, and hence continue to be unprofitable. The dairyman can ill afford now to retain a theoretically good cow which has recently aborted or had retained afterbirth. He knows very well that in nine cases out of ten she can not be profitable, and, under present conditions, is compelled to sell. The war has not directly made dairying unprofitable, but the war prices of food stuffs have so accentuated the difficulties of the dairyman that he is forced to yield.

In the past tuberculosis has played an important part in the economics of dairying and has been much in the public eye, but the sterility-abortion-calf scours group is collectively causing, and has long caused far greater losses than tuberculosis. The group of diseases probably adds one cent to the production cost of each quart of milk. It is to be hoped that the war conditions may serve to direct proper public attention to the enormous losses referable to this group of diseases and cause more vigorous and effective research work to be undertaken.

With the limited resources available, it has been our aim to study the subject of contagious abortion from every possible angle:

(1) We have examined the genital organs of more than five thousand cows, heifers, and bulls in abattoirs, and noted the diseased conditions.

(2) We have brought together at the college for study and illustration a very extensive collection of diseased genital organs of both sexes.

(3) We visit many of the leading herds of pedigreed dairy cattle in various parts of the United States and Canada, and examine clinically the genital organs of diseased animals. Animals deemed curable are given the best treatment we can devise to overcome sterility and prevent abortion, retained afterbirth, etc. We have observed as closely as possible the practical results of this work. If an animal seems hopeless we have endeavored to make post-mortem studies of the organs, in connection with her history.

(4) As far as possible there have been maintained correlative laboratory studies in bacteriology, pathology, and serology. Our opportunities in this direction have been very meagre. I have had neither time nor skill for this work, and what has been accomplished prior to the past year has been due chiefly to the hearty co-operation of colleagues, especially of Dr. C. P. Fitch of the Department of Bacteriology. A year ago, special provision was made for assistance in this part of the work, by appointing a laboratory worker with the grade of *Instructor*, essentially all of whose time was devoted to laboratory studies upon this disease. The position for the first year was filled, with great ability, by Dr. W. A. Hagan.

(5) For a number of years, a few experimental cattle have been kept, upon which detailed experimentation and close observations could be made. The number of such animals was seriously restricted and accommodations for their care wholly inadequate. In 1916 the Legislature, chiefly upon the representations of breeders of dairy cattle, appropriated fifteen thousand dollars for buildings, cattle, and sustenance, for the prosecution of this work. A substantial building, with good accommodations for thirty cattle, is now completed and occupied. This part of the work can now be greatly increased.

(6) We have endeavored to give due study to the problem of calf scours, calf septicaemia, and pneumonia, as an integral part of the sterility-abortion group.

By these means, it has been aimed to secure a broad and practical view of the problem, which might afford an adequate basis for recommending a practical plan for control. In the report of 1912 it was estimated that the monetary losses in the State of New York were not less than seven and one-half million dollars annually. Further study of the disease group convinces us that the estimate was too low. Since that time the disease has increased in virulence in the State. While the original estimate applied to the State of New York only, there was no intent to intimate or admit that New York suffers more or less than other states. Requests to attend and address various breeders', dairymen's, and veterinary societies and several colleges and universities have afforded unusual opportunities to learn of the conditions prevailing amongst cattle of other States. In conjunction with these meetings, numerous prominent herds of pedigreed dairy cattle have been visited and carefully examined. These opportunities for observation have made it perfectly clear that all parts of the country suffer alike. The losses are not so great, though by no means negligible, in beef as in dairy cattle. In large dairy herds, where most of our observations have been made, the disease, or group of diseases, is just as virulent in Massachusetts, Minnesota, and California as in New York. If the total losses from the diseases are less in California than in New York, it is because there are fewer dairy cows in large herds in California. The ratio of disease is the same. Neither is there any recognizable difference in breed. The total loss in New York is greatest in Holsteins — not because they suffer more than Jerseys or Brown Swiss, but because there are far more Holsteins. Animal for animal, the losses in other breeds are quite as great.

Observations upon the killing floors of abattoirs have shown that the frequency of marked disease of the genital organs follows the rule of prevalence of disease observed in herds. Severe disease of the genital organs of cows is commonest in the concentrated dairying regions, and least common in the beef-growing sections of the country.

Our studies of contagious abortion, as understood by us and outlined above, have led us to formulate recommendations for its control. Inevitably, the outlines have been of a very general character and have been subjected to numerous important amendments as research and experience have dictated. Our knowledge of contagious abortion, in all its complexities, is entirely too scant to enable us to lay down in detail permanent rules. The basis of our plan, as stated in 1912, has not been changed. We have consigned to a negligible or minor place:

(a) Isolation or quarantine of aborters.

(b) The administration of methylene blue, carbolic acid, and other drugs.

(c) Treatment by vaccination or serum.

(d) Disinfection of the gutter and stable.

The objections to quarantine have been considered on page 66. The chief objections to isolation are that it gives to the breeder a wholly false sense of value and fails utterly to check the disease, and is virtually always detrimental to the patient, because she is placed in poor quarters and neglected. If the aborter can be placed in a better stable and given better attention, isolation is advisable — not to eliminate immediately the danger to other animals, but because the better housing and care are better for the patient, for the owner, and finally, when the cow is returned to the herd, for the herd itself. Isolation must not be depended upon to prevent other pregnant cows in the stable from aborting or having retained afterbirth. If the isolation, or even the incineration of all observed aborters for one year or five years would stop or sensibly repress contagious abortion, it would richly repay the federal government to do this, but everyone of extensive experience knows perfectly well it would have no visible effect upon the prevalence of the disease.

No chronic infectious disease has ever been controlled by the administration of drugs. Perhaps the most notable effect ever obtained by drugs in a chronic malady is in syphilis, but that affects primarily the individual alone. The administration of salvarsan has not stopped syphilis, nor does it promise to do so. A drug or drugs may ultimately be found capable of modifying favorably or curing contagious abortion, but it is criminal folly

to stand idly by, awaiting the discovery, or even to dream that a drug will be found which will enable breeders and dairymen to ignore with impunity the fundamental laws of sex hygiene in cattle.

Vaccines, bacterins, and sera are interesting experimentally. We have tried the vaccines, or bacterins, as already recorded in our reports for 1911–1912 and 1915–1916; we have observed experiments with the newer proposition of hyper-infection of the non-pregnant animal; and we incorporate in this report a third plan — the administration of serum to pregnant and non-pregnant heifers. Like drugging, each of these plans is in the experimental stage, if not already discredited. At best, like drugs, they can never prove a panacea for the disloyalty of the breeder to fundamental rules of hygiene.

We hold that, so long as the infection is multiplying in the uterine cavity or the cervical canal of the cow, the disinfection of the gutter will not and can not control it. It can do no good to disinfect the gutter when it will be reinfected immediately by the uterine discharges from the diseased cow. If the genital canal has been disinfected so that no further infective discharges occur, it is useless to disinfect the gutter, as it is not infected. The same holds true when one attempts to disinfect the exterior of the buttocks, vulva, and tail. The disinfection can only be temporary, so long as the uterine disease in active form exists. When the internal disease is controlled, the external contamination ceases. This view does not affect the general problem of stable disinfection, but merely denies its power to affect favorably the infection of contagious abortion.

Our recommendations for the control of the infection are based on two fundamental principles.

(1) In breeding, mate only those animals, of each sex, whose genital organs are healthy.

(2) Guard the new-born calf, male or female, against infection through the milk or from diseased calves.

In order that a heifer or a cow may breed promptly, pass her period of pregnancy safely, and give birth physiologically to a sound calf, her genital organs must, first of all, be sound. She must have sound ovaries, oviducts, uterus, cervix, vagina, and

vulva. Unfortunately, even the genital organs of the heifer are not always sound. Before hygienic breeding can occur, the diseased genital organs must recover. It is essential that the soundness of the genital organs be determined *before, rather than after service*, because, according to a general law, the use of a diseased organ intensifies the disease. In infectious diseases, not only is the malady intensified, but it becomes increasingly dangerous to other contact animals. In infections of the genital organs, copulation tends always to intensify the disease present, harming the patient and increasing the peril to other animals. Consequently, wherever the rule can be applied it is desirable that the genital organs of each cow — and bull — should be examined thoroughly, and their freedom from recognizable disease made clear before the animal is permitted to copulate.

If contagious abortion is recognized as an endless chain of infection, transmitted frequently from dam to offspring, either in utero or shortly after birth through the medium of milk or other avenue, and later transmissible between the two sexes by coitus, the control of the infection involves the breaking, or rendering harmless, of the infection-cycle at some point, and thereafter guarding the non-infected or slightly infected animal against dangerous contamination.

In the present state of our knowledge, the infection, whether we regard it as pure or as mixed, works its chief havoc in the adult through the genital organs. In the new-born, it frequently induces mortal disease of the general system (septicaemia), of the alimentary tract (calf scours), or of the respiratory tract (pneumonia). Should the patient withstand the initial onslaught, the infection acquires a permanent habitat in the genital tract, where it persists in a dormant state to breeding age, when it becomes aroused and its ravages again become evident.

It is extremely difficult to outline a plan for the control of sterility, abortion, premature birth, metritis, and retained afterbirth in cows and of calf scours and pneumonia, and allied diseases, in young calves, which will be uniformly applicable in all herds and to all cattle. Some recommendations must be applied by the breeder or dairyman; some may be applied by the most highly intelligent breeders, in conjunction with a veterinarian;

and others can be applied only by the veterinarian. By no means are all veterinarians competent to apply many of the recommendations. The subject is new, and only a few of the practicing veterinarians have had valuable instruction or experience in this field.

Some of the recommendations are equally applicable to the handling of all dairy cattle; others can be afforded only in very valuable animals. The breeder or dairyman must decide, upon economic grounds, whether certain measures are applicable. *Whatever is done, the measures are to be regarded as an investment for the production of sound cattle, and the dividends are to be expected only after two or more years of intelligent and patient effort. The plan will not eliminate the disease, but merely bring it under control.* Dairy cattle and cattle foods have become so high-priced that the point has been approached, if not already reached, where no breeder or dairyman can afford to own a cow of such low dairy efficiency that her value will not repay giving her the best care possible.

The following plan is suggested, the details to be modified according to circumstances:

(1) Before breeding a cow — and if any serious question arises let the same rule apply to a heifer — the entire genital system should be thoroughly examined by a skilled veterinarian fully conversant with the diseases of the genital organs, in order to determine whether the animal is in good condition to conceive and is safe to breed. If disease is found in any part of the genital system, it should be cured before permitting the female to be served. If she is incurable, she should not be served, but should be sold for slaughter.

(2) When it has been determined, as nearly as is possible, that the genital organs are sound and the cow is ready to breed, she should be bred to a bull which is, so far as can be determined, sound. Prior to the service, the genital organs of the bull should be washed, for the protection of the cow, and after service washed again, for his own safety.

(3) After the cow has conceived, special precautions may be omitted until near the time of calving. The cow should be given good general care in order to maintain her health and vigor.

(4) A few days prior to calving, the cow should have a thorough bath over her entire body, with warm water and soap containing a fair amount of a reliable antiseptic. This is done in order to have her body surface clean, which will reduce as far as possible the danger to the new-born calf from infections borne upon the exterior of the cow.

(5) The cow should be given close attention for the first week after calving. She should not be milked during the first twenty-four hours. If the afterbirth is retained or if there is inflammation of the uterus without retained afterbirth, this should be given prompt and skilful attention — not only to reduce the danger of death, but to preserve as far as possible her dairying efficiency for the lactation period concerned, and more than all else to insure the promptness and safety of breeding at the next period.

(6) The calf should be removed immediately from the cow; the cow should not be permitted to lick the calf, and the calf not permitted to suck. It should be washed and dried, or dried without washing, with clean towels; its navel stump disinfected; and a high enema of salt solution (one ounce of salt to one gallon of warm water) given, in order thoroughly to empty the bowels. The enema should be repeated twice daily for two or three days. No food should be given during the first twenty-four hours. Then approximately one pound of clean milk for each fifty pounds of body weight should be given twice a day, increasing very gradually after four or five days, until the desired ration has been reached.

Considering these fundamental points at somewhat greater length:

In determining whether a cow is fit to breed, the veterinarian should palpate per rectum the ovaries. These are subject to a great variety of diseases, the principal of which are:

(a) a peculiar type of ovarian cyst causing nymphomania.

(b) cystic disease of the corpus luteum, preventing conception during the estrum involved.

(c) retained or hypertrophied corpus luteum, preventing estrum.

(d) adhesions of the ovary.

(a) That type of cystic disease of the ovary which produces nymphomania, or "bulling," appears to be due to cystic disease

of the ovisac, or Graafian follicle. In these cases, no corpus luteum is present; the cow bellows like a bull; attempts constantly to mount other cattle; and is generally erratic in her behavior; the ligaments of the pelvis become relaxed; the gait is unsteady; the bones of the pelvis slip upon each other, causing a cracking noise; and occasionally the pelvic bones are broken. The cow generally loses condition. Examined per rectum, the ovaries are found to contain cysts. Frequently only one ovary is involved; sometimes both. In some cases the cysts are single; in some multiple. Sometimes two or three cysts occur in the same ovary. They vary in size — sometimes only one-half to three-quarters of an inch in diameter and sometimes three to four inches. Sometimes these cysts are thin-walled and readily ruptured; sometimes their walls are thickened and the cysts may be ruptured only with great difficulty. In extreme cases they can not be ruptured by pressure through the vagina, but must be stabbed in order to evacuate their contents. Associated with the cystic disease of the ovaries — and possibly causing the ovarian disease — there is an enlargement and flaccidity of the uterus. Frequently the organ is two or three times its normal length, the vagina and vulva are also flaccid, and in some cases there is prolapse of the cervix.

The prognosis is fair. The outlook for recovery is greatly enhanced if the treatment is undertaken early. In old-standing cases, where the disease has existed for one or two years, the prognosis is bad, but not hopeless.

The treatment consists fundamentally of evacuating the cysts as rapidly as they form and disinfecting the uterus and cervix. Very thin-walled cysts which rupture easily may be ruptured by manual compression per rectum. If the cyst wall is at all firm, however, it is dangerous to attempt rupturing it in this manner. Instead, the ovary should be grasped per rectum with one hand and drawn backward above the vagina. The operator then inserts the other hand into the vagina, grasps the ovary, and applies the compression to it through the walls of the vagina. The vaginal walls are tough, and not readily injured if ordinary care is taken; the rectal wall is exceedingly fragile and readily damaged, or even ruptured. The compression of the cystic ovary through the vagina may be carried to the limit of the power of a strong man, providing

his finger-nails are well trimmed and care is taken to exert the pressure with the balls of the fingers — not with the nails. If these efforts fail, the ovary may be stabbed through the wall of the vagina and the contents allowed to escape.

The uterine cavity should be douched in the same manner as in chronic metritis not associated with cystic disease of the ovaries. If disease of the cervix is apparent, it should be handled the same as that described later, under "Cervicitis."

(b) *Cystic Disease of the Corpus Luteum.*— This consists of the formation of a cyst in the centre of the corpus luteum, or yellow body, which gradually enlarges, while the amount of lutein, or yellow tissue, gradually decreases. It begins as a very small cyst the size of a pinhead, and gradually increases in size, while the lutein tissue disappears, until there arises a cyst three-quarters to one inch in diameter, with a very small remnant of lutein tissue at some points upon the periphery. Finally all lutein tissue disappears, and apparently the cyst ruptures, after which the cow is again in estrum. So far as determined, the cystic disease of the corpus luteum has little significance beyond the one estrual period to which it belongs. Generally it appears to rupture within about three weeks, and disappear, a new ovisac ripens, and estrum recurs at the regular time. The only serious result of cystic corpus luteum known is that the condition prevents conception for the estrual period involved, in almost all cases. That is, the cystic degeneration of a corpus luteum is good evidence that disease existed in the precedent ovisac, which prevented fertilization of the ovum. Apparently the cystic corpus luteum is due to the presence of an infection and, the longer the infection is permitted to remain in the corpus luteum, the more probably it will affect the ovary unfavorably, causing similar disease in the next ovisac to ripen in the ovary involved.

Whenever cystic disease of the corpus luteum has advanced to a point where the cyst is three-eighths of an inch in diameter, it can generally be diagnosed by palpation per rectum. The corpus luteum is soft and fluctuating.

Ordinarily the prognosis is good, though in some cases the yellow body becomes cystic after each estrum. Since so far as we know a yellow body which is to become cystic can form only after

rupture of a diseased ovisac, it is possible — and appears to be true — that the ovary becomes so badly infected that no fertilizable ova escape from the ovary, and consequently the sterility must persist until a healthy ovisac ruptures and discharges a healthy egg. In a very few cases, in the abattoir, I have found a small cyst in the corpus luteum of a pregnant cow, but this is not the rule.

The treatment of cystic corpus luteum consists of pressing out the diseased yellow body. This should be done in the manner described for rupturing cysts. When compressed, the cyst in the corpus luteum usually ruptures first, and when the compression is continued the lutein tissue is finally detached and drops out into the hand.

(c) *Retained or Hypertrophied Corpus Luteum.*— When parturition is followed by pyometra, retained afterbirth, etc., the corpus luteum of pregnancy tends to sink deeply into the centre of the ovary and remain more or less permanently. Its presence interferes with the contraction of the uterus, and consequently with the expulsion of the diseased contents within the uterine cavity. The yellow body may persist for months, while the diseased uterus remains static or improves very slowly and the cow shows no signs of estrum. In such cases the dislodgment of the corpus luteum by compression through the vagina arouses the physiologic activity of the uterus and favors its restoration to health. If there is not much pus or other serious disease of the uterus, estrum ordinarily recurs within three to five days after the removal of the retained corpus luteum.

When desiccation, or mummification of the fetus occurs, the corpus luteum tends to sink into the centre of the ovary, as in pyometra, and remain month after month. The desiccated fetus remains in the uterus, and no estrum occurs. The mummy is so hard and dry, and the cervix is so thoroughly contracted, that mechanical removal from the uterus is dangerous and inadvisable. If however the corpus luteum can be reached and dislodged, the uterus contracts in from five to fifteen days and expels the mummy. Then the uterine cavity may be disinfected and the breeding powers of the cow restored.

In some cases a corpus luteum is normal in size, form and consistency, or slightly enlarged, and the uterus to all appearances

is comparatively sound, but the corpus luteum, for some unknown reason, remains permanently in the ovary, preventing estrum for months, and sometimes misleading the owner to believe that the animal is pregnant. In such cases, the dislodgment of the yellow body ordinarily causes a new ovisac to ripen and estrum to appear within three to five days.

Sometimes the corpus luteum is enormously hypertrophied or enlarged. Normally, this body is about one-half to three-quarters of an inch in diameter, but it may reach two inches or more. Frequently there is in the center of the corpus luteum a blood clot — sometimes merely the size of a pinhead, sometimes one and one-half to two inches in diameter. Sometimes the corpus luteum is virtually all blood clot. Such a corpus luteum as a rule finally becomes cystic, but it may persist for a long time, preventing estrum. It should be dislodged but in dislodging it the veterinarian should always be very careful, lest serious hemorrhage follow. After dislodging the corpus luteum, the veterinarian should compress the ovary in the hand for a few minutes, and then watch carefully to see that it does not bleed. A similar danger exists more rarely in the dislodgment of other types of yellow body, but generally the dislodgment is without material danger. However, it is well, as a general rule, to be careful in dislodging the corpus luteum, and in case of doubt to make sure that bleeding is not following the operation.

(d) *The Adherent Ovary.*— In numerous cases of inflammation of the oviducts or of the uterus, the infection passes out into the peritoneal cavity, around the ovary, and causes the gland to become adherent in the ovarian pocket of the broad ligament. In many cases the pavilion of the oviduct is closely adherent to the surface of the ovary. In any disease of the peritoneum in this region, as in tuberculosis, the peritoneal surface of the ovary is always liable to become adherent to the contiguous surfaces of the broad ligament and oviduct. Whenever this is recognized, the breeding life of the animal is at an end, so far as the ovary involved is concerned. If only one ovary is involved, the animal may breed upon the other side. There is nothing to do for adherent ovaries, except possibly in some cases where one ovary is adherent and the other is sound, and some disease persisting in

the adherent ovary is preventing estrum. In such cases it may be possible sometimes to remove the adherent ovary, thus destroying its inhibitory power upon the sound ovary, permitting estrum to occur and fertilization to take place.

The veterinarian needs determine also, by careful rectal exploration, whether the oviducts, or Fallopian tubes, are healthy or diseased. The ducts are subject to two different types of disease - - hydrosalpinx, or dropsy of the oviducts; and pyosalpinx, or pus in the oviducts. By rectal palpation, the diseased oviduct is found enlarged, and usually very hard. Sometimes it is adherent; sometimes free. The normal oviduct is barely palpable, as a very fine cord, about 1/32 inch in diameter. When diseased, it increases in size to one-half inch, or even more, in its transverse diameter. It becomes elongated also, and more intricately coiled than in health. It lies in front of the ovary and to the lateral side.

The cystic oviduct is very firm and rather uniform in the degree of enlargement. Frequently it is adherent to the surrounding parts. Sometimes the oviduct is quite free, so that it can be picked up and traced from end to end. Dropsy of the oviduct is bilateral in a majority of cases. If only one duct is involved, the animal may breed upon one side; if both ducts are involved, the animal is hopelessly sterile. This is one of the common causes of incurable sterility in heifers. In cows, it appears to follow retained afterbirth or metritis. It is not subject to treatment.

The second type of disease observed in the oviducts — pyosalpinx, or pus in the oviducts — except when due to tuberculosis, generally follows retained afterbirth or other form of severe metritis. When pyosalpinx is present without tuberculosis, the duct is not very large, as a general rule, but is hard and adherent. In many cases, instead of the duct enlarging generally, it yields at a certain point and an abscess forms. The abscess may be of any size from one or two inches to eight or ten inches in diameter. The ovary becomes lost in the mass. The abscess becomes adherent to various organs, and may rupture into the rectum, bladder, or elsewhere. Pyosalpinx and abscess of the oviduct are each incurable and render the animal permanently sterile upon the affected

side. If pus flows from the duct into the uterus, the animal is incurably sterile. The pus discharging into the uterine cavity prevents fertilization from the other ovary.

The uterus is subject to a great variety of diseases — or perhaps more properly to an exceedingly variable type of metritis, or inflammation of the uterus. There is every possible gradation, varying from a barely recognizable infection within the uterine cavity to an infection sufficiently virulent to cause complete necrosis of the entire organ. At what time infection becomes lodged in the uterus, we do not know. It has been found occasionally in the uterus of a new-born heifer calf. From clinical observations, it would appear that some of the virulent infections of new-born calves have a special tendency to invade and become fixed in the genital tract. Recent researches seem to show that one of the chief reasons, if not the great reason why abortion and retained afterbirth are so common in heifers in first pregnancy is because they become highly infected as young calves and the infection finds its way to the genital tract, to persist there in a dormant state until breeding age, when it becomes more or less aroused, to cause sterility, or, in pregnancy, increases in virulence, to cause abortion. The infection in the uterine cavity in the heifer, as in the adult cow, seems to persist chiefly in the cervical canal or at the cervical end of the uterus and in the apices of the horns. Such infection in the heifer does not at first cause any appreciable change in the uterus itself. If however the infection is sufficient to prevent impregnation, and copulation occurs frequently, the venereal excitement apparently intensifies the infection, and the uterus becomes enlarged and flaccid. That is, a definite chronic metritis, with atony of the uterus, occurs. This is usually associated with cervicitis.

The diagnosis of intra-uterine infection with mild metritis in the heifer is not very easy. It is usually to be based upon the slight enlargement of the uterus, with healthy ovaries, oviducts, and cervix. The outlook for recovery is good, if handled with reasonable promptness. If a heifer which has never conceived is neglected for one to three years, her outlook for breeding constantly decreases.

The treatment consists of the disinfection of the uterine cavity and of the cervical canal. Generally it is difficult to invade the

uterine cavity of the heifer. The cervicitis usually accompanying the chronic endometritis causes a swelling of the annular folds of mucous membrane in the cervix, distorting and compressing the cervical canal and rendering it very difficult to pass an instrument through into the uterine cavity. This causes the condition generally designated " closure of the womb." There is no closure, in fact, beyond the tight compression of the sides which renders the canal very narrow and also very crooked, so that it is difficult to follow, even with a small instrument. With care, as a rule, the veterinarian may introduce through the canal Palmer's uterine dilators, or a similar instrument, and gradually dilate the canal until a uterine catheter may be introduced and the cavity douched. Sometimes it is impossible to follow promptly the narrow, crooked canal with an instrument, and the effort needs be suspended, to be tried again at a later date. In such cases, if the veterinarian will dislodge the corpus luteum from the ovary which has last functioned, the heifer will be in estrum within from three to five days. At this time the canal is dilated and it becomes comparatively easy to introduce an instrument into the uterine cavity. The uterine cavity should then be thoroughly douched with a reliable antiseptic, such as two per cent Lugol's solution, after which in many cases a dram or two of iodoform suspended in oil may be introduced with benefit, and left in the uterine cavity. The treatment should be repeated about once a week for three or four weeks before attempting to breed the animal.

After the heifer becomes pregnant, if slight metritis or infection of the uterus exists, the metritis tends to increase throughout the pregnant state, especially when the infection is chiefly massed at the cervical, or posterior end of the uterine cavity. When located at the apices of the horns, the infection appears as a rule to be largely overcome, but leaves its trace in the form of the necrotic tip of the fetal sac, especially in the non-pregnant horn. When the infection is at the cervical end of the uterus, it does not reveal itself ordinarily during the continuation of pregnancy. If it is severe however, it may cause swelling of the udder, discharge from the vulva, and other symptons of approaching disaster. If the metritis has progressed less rapidly, premature birth may take place, or pregnancy may continue to full term, but the calving is

not normal. It may be decidedly slow, and on acount of the partial paralysis of the uterus the heifer or cow may require aid in the expulsion of the fetus. After the fetus is expelled, perhaps the afterbirth is retained. Then the symptoms of metritis, or infection in the uterus, become apparent.

If the fetus is alive and the infection in the uterus has been severe, there is great danger that the calf will suffer from septicaemia, calf scours, or pneumonia. The metritis at this period takes on a great variety of forms, depending upon the intensity. In some cases the metritis is so intense that the uterus becomes completely necrotic without the fetus having been expelled. The fetus becomes emphysematous and the cow or heifer quickly dies. If the fetus is expelled, either alive or dead, and the infection is intense, but short of actual necrosis of the uterus, the afterbirth, or placenta, is liable to be retained, to constitute the most dangerous type of metritis at this period.

Retained afterbirth always represents an intense and long-standing infection of the uterus, and nearly always a correspondingly virulent infection of the fetus. Often the fetus succumbs to the infection while yet immature and is expelled (abortion). Frequently the metritis induces the expulsion of the immature live fetus (premature birth). Most commonly, the pregnancy proceeds to full term, but the calf is almost certainly seriously infected.

If one will follow in detail until her next conception the treatment and progress of a cow which has suffered from metritis during pregnancy, and which after the expulsion of the fetus has retained afterbirth, it will afford a valuable illustration of the plan for handling a highly important phase of the entire problem.

Retained afterbirth in cows represents a common type of "contagious abortion". It may be observed subsequent to abortion, premature birth, or birth at full term. It is a disease which becomes established *during, not after pregnancy.* Retained afterbirth is not observable during pregnancy, and is rendered visible only after the fetus has been expelled.

Many doubt that retained afterbirth is always or generally contagious abortion, but the controversy is academic, not practical. Retained afterbirth, strictly defined, is placentitis, or cotyledonitis. The placentules, or cotyledons, become inflamed, the walls

between the crypts become engorged and swollen, and the tufts of the chorion, also engorged, become incarcerated within the placental crypts. The placentitis is due to an infection which has advanced from the cervical canal or the cervical end of the uterus, forward toward its ovarian end. Usually it advances slowly, first causing disease of the cotyledons at the cervical end, and gradually involving one after another. If but a few cotyledons at the vaginal end of the uterus are badly diseased, pregnancy continues, but an irritation exists in the posterior portion of the uterus, which may cause the expulsion of the fetus, mature or immature. When placentitis becomes established, the capillaries in the walls of the placental crypts and the tufts of the chorion become blocked, as in inflammation of other tissues, the placental circulation ceases, and the carrying of nutrition to and waste products from the fetus is stopped. If a sufficient number of cotyledons is involved, nutrition is cut off, and the fetus perishes, or the fetus may perish because the attendant infection in the utero-chorionic space has penetrated the amniotic fluid, has been swallowed by the fetus, and has induced a mortal infection. The fetus may then be expelled: the cow aborts. The abortion is caused by the infection existing within the uterine cavity. The infection is presumably transferrable to the uteri of other animals of the same species and capable of inducing abortion in these. It is not essential to prove or disprove that retained afterbirth is generally or always due to the bacillus abortus of Bang. The really important point is to know that the abortion is due to an infection or contagion, and to appreciate keenly the fact that the contagion has a peril for the dam and the fetus, and, through these, for other susceptible animals.

Retained afterbirth can not be positively defined. Many do not regard an afterbirth as retained unless it remains in the uterus ten or twelve hours after the termination of pregnancy. In the researches of this department, where accurate observations have been made, it has been found that when the uterus is sound, so far as we can recognize, the fetal membranes are ordinarily expelled within one and one-half hours after the expulsion of the fetus. We consider that when an afterbirth remains in the uterus more than one and one-half hours after the expulsion of the fetus the condition is pathologic.

The symptoms of retained afterbirth are ordinarily too obvious to require relation. Depending upon the degree and character of the metritis, there may be a great variety of symptoms, but these are the symptoms of the metritis and the placentitis, not of the retained afterbirth. Except for the presence of the fetal membranes themselves, all phenomena may be, and commonly are present in metritis unaccompanied by retained membranes. The retention of the membranes is striking and indubitable evidence of the presence of metritis of a severe character. It is a very perilous complication of an old-standing metritis, especially involving the placentules, or cotyledons.

The importance of this type of metritis has generally been grossly underestimated. Veterinarians and breeders frequently think of it as a common and unimportant occurrence, which may lower temporarily the condition and efficiency of the patient. This is a most unfortunate conception, unsupported by careful clinical observation. Retained afterbirth is one of the most unfavorable phenomena in the whole complex group of disasters falling within the scope of the infection of contagious abortion.

The mortality from retained afterbirth, while not representing the major loss, is by no means trivial. On the whole, it probably amounts to three to five per cent. At times it breaks upon a herd as a furious storm, with an appalling mortality. I observed one outbreak in a stable of about sixty breeding females, where twenty per cent. succumbed to the virulent infection and another twenty per cent. of the herd eventually went to the shambles because of incurable sterility. The milk production for the year was well-nigh ruined, the calves largely succumbed to septicaemia, scours and pneumonia, and the herd was virtually wrecked for a long time. Nothing could stay the disaster. At the close of parturition, the metritis was intense, the uterus already paretic, the parturition slow and requiring aid, and, once the genital canal had been dilated and freely opened by parturition, the infection ran riot, and all efforts at control were fruitless. Such outbreaks are not rare. They are merely an intensification, a massing of an infection common in dairy herds. In almost every dairy, now and then, an isolated case of this character occurs.

Retained afterbirth is the commonest forerunner of temporary or permanent sterility, and almost universally it lowers, and fre-

quently ruins the milk production for the entire lactation period. The advice of veterinarians regarding its handling is exceedingly conflicting at every point. Owners and herdsmen frequently have fixed notions regarding the proper course to pursue, and fail to cooperate with the veterinarian. Many insist upon the manual removal of the afterbirth; many others demand as emphatically that it be left alone. There is so much controversy over the question that when one speaks of manual removal it is always difficult, and frequently impossible, to know what is meant.

The fetal membranes, whether expelled or removed manually, become separated from the uterus by two distinct methods:

(1) *Physiologically.*— The chorionic tufts drop spontaneously out of the placental crypts. As soon as the fetus has been expelled and the navel cord ruptured, circulation through the chorionic placentae ceases, the blood within the capillaries, which constitutes the chief volume of the chorionic tufts, escapes through the ruptured ends of the umbilic veins, and the tufts collapse. The physiologic attraction between fetal and maternal placental structures is destroyed. There is no continuity of tissue at any time. When the physiologic attraction ceases and the capillaries in the tufts collapse, the tufts fall away from the crypts in the cotyledons and lie free within the uterus. Later the contractions of the uterus press the membranes out into and through the vagina. The placental crypts are left denuded of protective epithelium.

(2) *Pathologically.*— Some of the capillaries in the placental crypt walls are engorged, swollen, and perhaps permanently blocked by thrombi. The capillaries in the chorionic tufts are similarly involved. As a consequence, the chorionic tufts are incarcerated within the crypts and more or less firmly held. After the allantoic vessels have ruptured, no material increase of volume of the chorionic tufts may occur. No additions of blood or lymph may occur. Perhaps a few leucocytes may penetrate them; probably not. The entire mass of fetal tissues is necrotic and passive. If the inflammation is not too severe, the weight of the membrane mass may gradually pull away the chorionic tufts and the afterbirth come away in three to ten or more hours. After the rupture of the allantoic vessels, even though their capillaries in the chorionic tufts are occluded by thrombi, portions of the thrombi

may be gradually pressed out by uterine contractions, and the volume of the tufts thereby decreased and permitted to drop away. Every part may undergo decomposition, and first of all the chorionic tufts within the placental crypts. If the crypt walls remain intact, the chorionic tufts may be expelled by serum poured out from the crypt walls. The placental tufts may more or less completely decompose and the remnant be pushed out after a lapse of some days, leaving the crypts filled with pus and inflammatory debris.

The incarceration of the chorionic tufts may be irreducible and the placental portion of the cotyledon become necrotic. The fetal and maternal tissues then usually become inseparable, and the afterbirth alone can neither be expelled by the uterus nor detached by the surgeon. The cotyledon must then become more or less completely necrotic and slough away from the uterus (if the uterus too does not become necrotic) and the fetal membranes expelled, or lie detached in the uterine cavity, the necrotic cotyledons detached from the uterus, and inseparably attached to the fetal chorion. The chorion does not always remain attached to the necrotic cotyledons, but in some cases the membranes become detached and are expelled, leaving behind the badly infected cotyledons, which may be, or later become necrotic. Then there exist two phases of expulsion of necrotic debris — first, the fetal membranes in a mass; and, secondly, the necrotic cotyledons one by one.

No time is fixed for the completion of either of these processes. When the fetus is emphysematous, the cotyledons may have all sloughed away before the fetal cadaver has been removed, or the membranes may remain irremovably incarcerated and the cotyledons not yet sloughed off after two weeks or more. Between the extreme degrees of retained placenta and the physiologic expulsion of the membranes, there is every conceivable variation.

The metritis causing retained fetal membranes becomes established during pregnancy, perhaps essentially always very early in pregnancy. If it progresses rapidly and causes the death and expulsion of the fetus prior to the completion of the fifth month of pregnancy, the chorionic tufts are so short and simple that they do not become incarcerated. If the metritis is slower in its action and the pregnancy continues into the eighth month of pregnancy,

whether the fetus is expelled alive or dead, retention of the fetal membranes of a very vicious character is highly probable. When severe metritis exists at full term, the chorionic tufts, being more fully developed, tend to cause an even more severe retention of membranes than when the pregnancy terminates prematurely.

The metritis, with the placentitis, or cotyledonitis, causing the retention of the membranes, regularly begins at the vaginal end of the uterus, and generally advances slowly up to the termination of pregnancy. After the pregnancy has terminated, and the genital passages are dilated and their walls suffering more or less seriously from parturient contusions and abrasions, the infection spreads with great rapidity to the ovarian end of the uterus. Thus it is commonly found in retained fetal membranes that, when examined shortly after the expulsion of a living fetus, the chorion has already separated from some of the cotyledons at the cervical end. Slightly further forward, the chorionic placentae may be detachable from the uterus. Beyond, there is another group of swollen, hard, enormously enlarged cotyledons not yet wholly necrotic, and the chorionic tufts so firmly incarcerated that their withdrawal is impossible. Further beyond, the inflammation gradually decreases, until at the ovarian end of the uterus some cotyledons may be healthy, or nearly so. Hence it is a common clinical experience that the first six to ten cotyledons at the cervical end, in each row, may be detached or detachable, while the attachments of the membranes to the five or six cotyledons at the ovarian end may be immovably incarcerated. Last to become involved, the cotyledons at the ovarian end are last to release the chorion or to become necrotic and slough away.

The infection, the placentitis which causes retained fetal membranes, never begins at the ovarian end of the uterus. Metritis with placentitis beginning at the ovarian end of the uterus is common enough — in fact, is to a limited degree well-nigh universal in cows, as well as in other ruminants and in swine, but it is incapable, apparently, of causing retained fetal membranes or abortion. The necrotic tip of the fetal membranes in these animals appears to be due to a distinctly pathologic process, so common as to be designated *normal*, though there is excellent macroscopic evidence of a limited local metritis and choriontitis. Often in the

cow this metritis involves the entire non-gravid horn, so that it is wholly devoid of cotyledons. The fetal sac within the non-gravid horn is wholly necrotic in such cases, and there is no recognizable tendency to abort or to suffer from retained fetal membranes.

In the present state of our knowledge, it appears that an infection within the pregnant uterus may cause incarceration of the fetal membranes only when invading the uterus from the cervical canal. It appears also that, in order for retention of the afterbirth to occur, the uterine seal must have been destroyed or damaged to such a degree as to admit some oxygen ere the infection may accomplish the particular type of disease. The infection advancing from the ovarian end of the uterus is strictly anaerobic; the infection causing retained placenta, which advances from the cervical canal, is aerobic, with an extremely limited oxygen supply. As the oxygen supply increases, the virulence of the infection mounts, and when the uterine seal becomes completely destroyed pathogenic organisms requiring greater oxygen supply may invade the uterus, to complicate and further intensify the virulence.

In the handling of retained afterbirth, the veterinarian can not alter the line of progress of the disease. He may modify its intensity and more or less ameliorate its devastation. He may minimize greatly the dangers besetting the patient.

The greatest controversy about the handling of the condition is waged over the question of the manual removal of the membranes and when, if at all, the removal should occur. The controversy is based largely upon ignorance of the pathologic conditions present. The fetal membranes, immediately the navel cord has parted, constitute a necrotic mass. With the infection causing the retention already long present in the utero-chorionic space, ready to be fanned to a flame by the greater ingress of oxygen following parturition, and the open avenue for a new invasion from the vagina or from the exterior, the necrotic mass of membranes soon becomes putrid. The condition then facing the veterinarian is a great mass of rapidly decomposing necrotic tissue, held within a diseased hollow organ, the protective epithelium of which has been largely destroyed or seriously damaged. Practically, there is a great necrotic, decomposing mass of tissue, lying in an enormous wound. Is it desirable that this putrid mass be

removed? The fundamental principles of modern surgery admit of but one reply. Unquestionably, the removal of this putrescent mass is highly desirable. Unfortunately, there are many highly desirable operations which can not be performed, amongst which many cases of retained afterbirth constitute very marked and discouraging illustrations. When the cotyledon is intensely inflamed, swollen, enlarged, and hard, the placenta can not be detached. One may tear through the chorion about the periphery of the cotyledon, leaving the chorionic placenta attached to the cotyledon and the necrotic chorionic tufts still projecting their full length into the deep crypts. That is not removing the placenta; it is merely an ignorant or criminal subterfuge. The patient is only placed in greater peril by such malpractice. The human gynecologist can invade the short, broad uterus of woman and curette away the incarcerated fetal membranes, with the incurably or dangerously infected placental tissues, but no such possibility exists for the veterinarian. The hundred pedunculated cotyledons of the cow offer no field for curetting. If the cotyledon is wholly necrotic and the gangrene is limited by a line of demarcation through the pedicle, the veterinarian may have an advantage over his human confrere, and may simply detach the necrotic mass. Until he may detach the necrotic cotyledon with reasonable safety, he is helpless. The chorion can not be separated from the cotyledon, the cotyledon can not be safely separated from the uterus, and the veterinarian is compelled to wait. In the meantime, the patient may — and too frequently does — die.

The surgical impossibility of detaching the chorion from the cotyledons is not the only insurmountable barrier to the manual removal of the fetal membranes. There is always some degree of uterine inertia in a cow which aborts, calves prematurely, or calves at full term and has retained afterbirth. At full term, the uterus is five to six feet long, and in retained afterbirth, with the inertia due to the metritis, the uterus may contract very poorly and may remain almost its full preparturient length for hours or days after the close of pregnancy. Occasionally one hears of a man whose arms are so long that he can reach the ovarian end of the uterus. If the uterus is well contracted, a man with short arms can reach the ovarian end readily, but such a cow does not have retained afterbirth. If the uterus contracts little or not at

all, no man can reach the ovarian end. In unicornual twins, the two fetuses, each having a body three to four feet long, lie in the uterus, head to head or tail to tail, necessitating the elongation of the gravid horn to a minimum of eight feet. It is idle to talk about the manual removal of the fetal membranes in such a case until the uterus is well contracted — and that may occur in a day, a week, or never.

Stated in the simplest terms, the membranes should be removed whenever they can be entirely removed — whenever each of the chorionic tufts can be completely withdrawn from the crypts and the tufts and crypts each remain intact. When the cotyledons have sloughed from the uterus, or are necrotic and readily detached without causing irritation, pain, or hemorrhage, the fetal membranes, with the necrotic placentae, should be removed. The great trouble is to have people realize these fundamental principles. The owner calls the veterinarian to "remove the afterbirth," and removed it must be if it kills the cow. Sometimes the veterinarian takes the stand that he must remove the afterbirth, or the cow will die. That is not so frequently correct as the counter-view that, if he persists in his efforts to remove a badly incarcerated afterbirth, the cow will in all probability die. An attempt to remove an irremovable afterbirth is one of the very worst types of malpractice, and is the foundation for a deeply rooted prejudice against its manual removal.

When the membranes can not be completely removed, when the chorionic tufts can not be withdrawn from the placental crypts completely and without mutilating them, and when the cotyledons have not sloughed off or are not ready to drop off, and their removal thereby indicated, the veterinarian should await the eventual separation of the membranes by suppuration or other processes within the crypts, or the final sloughing of the cotyledons. In the meantime, the progress should be observed by frequent examinations and whenever practicable the membranes removed. The veterinarian should not tear the membranes in two and remove only a part, permitting the remnant to drop back into the uterus. This complicates the situation. The uterus is inert, while the cervix retains its contractile power. The cervix does not contract completely while a foreign body is in its canal.

If portions of the membranes project through the cervix into the vagina, they serve the useful purpose of keeping the cervical canal open, so that the operator's hand may usually be introduced at will or the membranes may escape. The weight of the membranes dragging gently upon the incarcerated tufts tends constantly to detach them.

While awaiting the detachment and removal of the membranes, their putrefaction should be retarded so far as practicable, in order to lessen the amount of bacterial irritation and of the resorption of bacterial products by the uterus. It has been proposed to douche the uterine cavity in order to check putrefaction. This is a gross misconception. The uterine cavity of the cow can not, by any means at present known, be douched while the fetal membranes are retained. When the operator carries a douche tube in, neither his hand, the tube, nor the liquid comes in contact with the uterus, except in a small denuded area of varying size at the cervical end. Beyond, the uterus constitutes a vast cavity, lined and screened everywhere by the detained fetal membranes. Between the uterine walls and the fetal membranes, are about one hundred cotyledons, arranged in two parallel rows on either side of the body and of each horn. The cotyledonal stalks are ordinarily one to one and one-half inch long. The cotyledons form a vast colonnade between the inner surface of the uterus and outer surface of the chorion. The surface of the uterus, at the close of pregnancy, has an area of about fifteen square feet. Between the uterus and the chorion, is a great complex cavity, crossed by the cotyledonal stalks. In retained placenta, this utero-chorionic space contains pus, "the exudate of contagious abortion," and tissue debris. The veterinarian has not yet devised a successful plan for invading the utero-chorionic cavity. The putrefying contents, in immediate contact with the uterus, defy all efforts at removal, until the retained membranes are out of the way.

The veterinarian can readily introduce his hand into the fetal sac through the parturient rent at the cervical end. Into this sac, he may carry a douching tube and introduce liquids into the fetal sac, but in many cases he can not douche the sac. The fetal sac is dead, and had no contractile or expelling power when alive. Whether one attempt to douche by means of a double tube or by siphoning, the membranes may persistently block the tube and

prevent any outflow. The cavity of the chorion is strewn here and there with the vessels of the umbilic cord, and the ruptured and shredded amnion hangs free in the cavity. These and the chorion itself tend to be sucked into the outflow and prevent the exit of fluids. In retained membranes following unicornual twins, with a uterine cavity possibly eight feet long, the uterus must be doubled upon itself, become kinked, and the outflow of liquids be prevented. In many cases of retained fetal membranes, the cervix is constricted — that is, it is normal — while the uterine walls are paretic. If a tube is inserted through the moderately constricted cervical canal, even though it passes readily, and fluid is introduced under the pressure of a pump, or even under gravity with a column of liquid five feet high, and the liquid does not return promptly, the uterus ruptures. Twice I had this occur, followed quickly by death, before I appreciated the danger of using a pump, though in each case the termination of pregnancy had been quite recent and there was abundant room to pass one or two fingers alongside the tube through the cervical canal. Similar disasters have occurred in the practice of many veterinarians. The danger does not consist entirely of probable rupture of the uterus. When several gallons of liquid are introduced into the paretic uterine cavity, and not expelled or siphoned out, the mechanical weight is a great peril to the organ. The paralysis already present is greatly increased by the weight of the fluid and the distention of the uterus. It needs be clearly understood that pouring a large volume of fluid into the chorionic cavity is not douching the uterus, and if the fluid is not expelled or siphoned out, or otherwise removed, even the comparatively useless expedient of douching the chorionic cavity has not occurred.

The posterior portion of the uterine cavity may be douched and the douching fluid come in direct contact with all that portion from which the afterbirth has become detached. However, it can not be douched with antiseptics, without serious injury. The denuded, highly inflamed mucosa will not tolerate any known solution having marked bactericidal power. Reliance must be placed upon a neutral liquid, as physiologic salt solution, to induce partial mechanical disinfection through washing. Not only are many bacteria washed away, but also bacterial products and tissue debris. The effort at douching must be limited to those cases in

which the fluid can be wholly removed. The uterus must contract and expel the fluid, it must be siphoned out, it must be pressed out by the operator exerting pressure upon the organ per rectum, or ladled out with his hand, or it must be removed by other means. It can remain only with great peril.

Novel methods for removing the placenta are now and then proposed. For a time hydrogen peroxide was hailed as a solution of the whole problem. In some cases it appeared efficient. Perhaps they were cases in which the membranes were ready to drop away. The pathology of retained fetal membranes is such that it is not apparent how hydrogen peroxide could favorably affect it. The difficulty is within the placental crypts. The chorionic tufts are necrotic and the crypt walls likewise undergo more or less complete necrosis, so that the entire placental mass, fetal and maternal, frequently becomes necrotic. It is not to be assumed that peroxide of hydrogen would penetrate this great mass, two to six inches in diameter, and bring about a separation between the fetal and the maternal tissues. It is neither necessary nor highly desirable that such separation occur, if the maternal tissue is necrotic.

More recently it has been asserted that the chorionic tufts may be forced out of the placental crypts by injecting salt solution into the chorionic capillaries through the umbilic arteries. If the capillaries in the chorionic tufts were not thrombotic, so that blood or other liquid could not enter, the incarceration would not occur. If the chorionic capillaries were open for the passage of fluids, their distention would increase, not decrease, the incarceration. There is no logical basis for hoping that this or any other plan can directly cause or hasten the detachment of the diseased membranes. Like other inflammations, the disease must pursue, within certain limits of variation, a prescribed course, dependent upon the extent and virulence of the infection and the power of resistance in the tissues involved.

When the membranes are irremovable, the rapidity of their decomposition and the formation of bacterial poisons consequent upon the putrefaction may be somewhat limited by introducing into the ovarian end of the chorionic sac, as far as possible, a feebly soluble, virtually non-irritant antiseptic. Amongst these,

iodoform occupies a prominent place. It may be applied as a powder, enclosed in a gelatin capsule, or it may be suspended in a neutral oil, as olive oil or mineral oil. When introduced into the chorionic cavity, it may be used virtually as one pleases, as its absorption is almost negligible. One ounce, and even more, appears to be uniformly safe. It has been claimed that, if the gelatin capsule is not broken and the iodoform emptied, the capsule may become encased in a layer of mucus and not dissolve. This is probably an error. Introduced into the uterus, the capsule may become coated with mucus, but there is no mucus or other substance within the chorionic cavity which would probably encase the capsule. The iodoform may be combined with an equal part of boric acid and with a small amount of thymol. It is probably best to introduce the iodoform suspended in oil. The distribution is more even, and the oil soothes where it comes in contact with living tissues. The veterinarian should watch the afterbirth, testing its detachability now and then. After three to five days, if the membranes are not yet out, the iodoform should be repeated. After the fetal membranes have been expelled or removed, the veterinarian may deal more directly with the metritis present. The metritis differs in no essential respect from the metritis without retention of the membranes. When the fetal membranes are expelled promptly and metritis is present, there occurs frequently, from the uterus, in from one to seven days a bright scarlet exudate mixed with yellowish flakes — hemorrhage with necrotic tissue particles. Frequently this exudate, emanating from the utero-chorionic space, precedes the dead or living fetus, or may follow it immediately, but if the membranes are retained the exudate soon ceases to escape in recognizable form. It becomes mixed with fragments of the decomposing membranes, and soon with pus. Largely it is detained in the utero-chorionic space, since the uterus is too inert to bring about expulsion. Hence, if the fetal membranes are long retained, the discharge characteristic of the early post-pregnant stage of metritis without retention of the membranes may pass unobserved because it is hidden away behind the membranes.

When the retained membranes have been expelled or removed, the metritis becomes in all essentials identical with the metritis

unaccompanied by such retention, and from this point the two types are to be considered as one. Whether the fetal membranes have or have not been retained, the denuded uterine mucosa will not tolerate active antiseptics, but it may be freely and effectively douched with physiologic saline solution, and all the fluid can be siphoned out. Sometimes necrotic detached cotyledons obstruct the outflow, but are removable. A large metal uterine catheter will permit large placental fragments to be washed out. If the cervical canal is not too constricted, the detached cotyledons may be pressed out alongside the douching tube, by exerting manual pressure upon the uterus from the rectum. The most complete douching of the uterus is not accomplished by douching tubes, but by means of a single pure gum tube, used alternately as inflow and siphon. While the cervical canal is freely open, the single pure gum horse stomach tube, with an irrigator of several gallons' capacity, is the best apparatus available, but its distal end is not as good as the smaller horse catheter, with its two lateral fenestra. The single terminal opening in the stomach tube tends constantly to suck in the uterine wall and stop the flow. When the cervical canal becomes quite narrow, the catheter is best, but no large masses of necrotic tissue can pass through it. With either rubber tube, the process is essentially the same. The distal end of the tube is pushed into the apex of one horn. Saline solution is introduced to dilute the excretions and debris present.

When a sufficient amount has been introduced, the outer end of the tube is to be detached and lowered to the floor, in order that it may act as a siphon. The pus and tissue debris lie mostly in the apex of the horns, which is the lowest part when the animal is standing. Usually the fluids drag the uterus down, and when the siphon is started the end is some distance posterior to the apex of the horn and the lighter portions of the contents flow out first; then, as the uterus is emptied of its load, it shortens, the tube reaches into the apex of the horn, and the heavier pus and debris flow out. In many cases when the uterus is well emptied, it begins to contract and relax alternately under the suction of the siphon; the outflow of contents alternately ceases and resumes. The operation serves as a form of massage or serves the purposes of electrical stimulus. The siphon process, when the distal end is passed into

the apex of the horn, empties it completely except in the narrowest part, where it is succeeded by the oviduct. At this point the douching can not be made perfect. Each horn should be douched. The metal douching tube may generally be passed into either horn, simply by turning the catheter in the proper direction. When the cervical canal is freely open, the gum tube may be carried into the desired horn with the hand, or may be directed by the hand from the rectum.

When the horse catheter is to be used, it is best introduced by inserting the tip of Palmer's uterine dilator or of a sound of similar form and size into the distal fenestrum of the catheter and pushing the catheter through the cervix with the metal instrument. As the catheter enters the uterus, it may be directed into the right or left horn with the dilators. The alternating introduction and siphoning out of the saline solution should be continued until the siphoned fluid is clean from both horns. The complete emptying of the uterus of pus, debris, and saline solution may be favored by manual compression and massage of the uterus from the rectum.

After the douching of the uterus has been completed, the metritis may be greatly benefited by the introduction of iodoform. The conditions have definitely changed however. Instead of the drug coming in contact chiefly or only with the necrotic chorion, it now comes in direct contact with the uterus. Unless the uterus is necrotic, in which case the iodoform is powerless to do good or harm, it is absorbed rapidly, and may induce iodoform poisoning in twenty to thirty minutes. I have seen a cow salivate freely and mucus flow abundantly from the nostrils after introducing less than one ounce, suspended in oil. It did no harm. The iodoform passes quickly to the udder also, so that within fifteen to thirty minutes the milk is repulsively tainted from the drug, and may remain tainted for seven to fifteen days. When iodoform is used in the uterus of a dairy cow, the milk should under no consideration be permitted to go to market until careful tasting gives assurance that the taint has disappeared. Since the healthfulness of the milk is in no way involved, it may be fed to calves. Douching the uterus with warm saline solution may be repeated as frequently as once daily, until the metritis is under control, followed

by the introduction of small amounts of iodoform in oil. To the iodoform oil, there may be added boric acid or thymol, if desired.

In a few days the uterus becomes more tolerant, and moderately efficient antiseptics may be used, but dependence must still be placed largely upon mechanical cleansing by washing. The chlorine-bearing solutions, as Dakin's solution, and the substitutes therefor, as chlorazene, appear to be highly valuable, but must be used cautiously. Chlorazene may usually be used in about 0.4 per cent. The compound iodine (Lugol's) solution may be used in 0.25 per cent., and cautiously increased in strength. Carbolic acid, lysol, creolin, and related compounds are too irritant, and should not be used. Corrosive sublimate is entirely too irritating.

As the disease progresses, the uterus becomes increasingly tolerant, and in old-standing metritis may be douched with as high as two per cent. Lugol's solution. If so strong a disinfectant be introduced, it must be completely expelled, pressed out by massage per rectum, or douched out with saline solution. If not expelled, its prolonged presence in the uterus irritates, and eventually intensifies the disease. Even a neutral fluid like saline solution must not be left in the uterus in large amount. When the cervix is well contracted and the uterus is small, a few ounces of neutral liquid injected into the uterus by gravity, under a column of water 20 to 36 inches high, if it does not escape, causes colic. At first the uterus may relax and become quite dilated from the pressure of fluid or from inflation due to an inrush of air through the tube. After a time, the uterine walls contract and, failing to expel the liquid or air, are seized with tonic spasms which induce severe pain. The cow kicks at her belly, switches her tail, lies down and gets up frequently. The douching of the uterus should be continued at varying intervals of not less than once a week, until the formation of pus or other exudate ceases. When active suppuration has ceased, the use of iodoform should ordinarily be discontinued, and wholly replaced by such disinfectants as Lugol's solution, chlorine-bearing drugs, or other disinfectants which do not taint the milk.

Not all cases of metritis are curable. Many cases assume a peculiarly virulent character: the uterus becomes gangrenous, the temperature vacillates, the eyes are sunken and listless, a profuse

discharge of muco-pus occurs from the nostrils and eyes, a severe diarrhea is present, the excreta are black and watery. Usually the animal succumbs in five to fifteen days, but may partially recover, though improvement is very slow. Pelvic adhesions are generally extreme. Incurable sterility is present. Frequently the animal succumbs finally to pyemic abscesses of the liver, spleen, articulations, or other part. In less severe cases where the cotyledons become gangrenous, the recovery may be quite complete and the breeding powers retained, but when the uterine mucosa is entirely destroyed the breeding life of the animal is closed, though general recovery may follow and the animal become fit for beef. When the afterbirth is retained and the cotyledons slough away, the necrosis in some cases involves the entire mucous membrane of the uterus, which sloughs away entirely, leaving a fistulous tract instead of the uterine cavity. Such a uterus sometimes appears normal upon palpation. If a catheter is introduced into it, a little pus may return with the douche, or the douche may come out clear. In most cases of necrosis of the uterine mucosa, the uterus becomes sclerotic and remains so permanently. Since in either case the outlook for the breeding of the animal is hopeless, she should be destroyed. In all cases where the metritis retains its purulent character and the pyometra can not be overcome within a reasonable length of time — say four to eight weeks — it is generally necessary to destroy the animal. Should suppuration cease and the uterus return to normal, the animal should have six to ten weeks of rest in order to permit the uterus to regain its tone, after which the uterine cavity should again be douched, partly in order to determine the exact condition of its cavity and partly to make doubly sure of the disinfection. If no pus is found in the cavity, the animal may be bred.

In all cases of persistent pyometra, the ovaries should be examined very carefully for the presence of a retained corpus luteum. If this is present, it should be dislodged as promptly as possible, because its removal destroys the inhibitory power which it has been exerting, and stimulates the uterus, causing œstrum to occur, with tonic contraction of the uterine walls and a thorough emptying of the uterine cavity.

In all cases of persistent purulent metritis, the veterinarian

should, by every means at his command, exclude the possibility of uterine tuberculosis. This is described in another section of this report, "Tuberculosis of the Genital Organs of Cattle."

Besides a collection of pus in the uterus, there occurs sometimes an accumulation of lymph or mucus in the uterine cavity. In very rare cases, this is due to some disease of the uterine wall, but as a general rule to disease of the cervix. The cervix becomes inflamed, especially the bases of the large annular rings in the cervical mucosa, which, pressing firmly together, bend the cervical canal abruptly, in variable directions, and so press upon the cervical canal that the menstrual debris forming within the uterus at the time of estrum can not escape. Remaining imprisoned, its volume is gradually augmented, frequently reaching two to four quarts, or even more. It then resembles pregnancy very strongly, but as a general rule the uterus is not so firmly distended and the two horns are approximately equal in size. In pregnancy, on the other hand, except in case of bi-cornual twins, the pregnant horn is very much larger than the non-pregnant. Retention of menstrual fluid within the uterus is to be treated by overcoming the difficulty in the cervix. The cervical canal must be entered carefully with dilators and sufficiently dilated to permit the introduction of a uterine catheter, through which the retained fluid is caused to escape. The uterine cavity must then be kept free from accumulation by repeated dilations of the cervical canal and the overcoming of any inflammation existing within it.

Cervicitis is one of the commonest and most serious types of disease of the genital canal in the cow. It is one of the commonest causes of sterility in heifers. A very large proportion of cows suffer more or less from inflammation of the cervical mucosa. As stated in the preceding paragraph, when the inflammation involves the annular rings of the mucosa, their bases become much swollen and sclerotic. Sometimes the bases of the annular folds become great tumor-like masses ½ to 1 inch in diameter, protruding into the cervical canal from one side and pushing the canal before it, in a manner to cause a very acute bend. In the early days of veterinary science, this condition was designated "closure of the womb." The idea was that there was an actual closure of the cervical canal. This is not true. It is only in very rare cases that

the canal becomes closed in such a manner that the fluids forming in the uterus do not readily escape through it. In those rare cases, the uterus becomes distended with the menstrual debris, as mentioned in the preceding paragraph. At the vaginal end of the cervix, a very interesting change occurs in the arrangement of the mucosa. In the heifer, the vaginal end of the cervix constitutes a very sharp cone pointing backward toward the vulva. At the apex of this sharp cone is the os uteri externum. After the animal has passed through one or more pregnancies, this original cone disappears. The first annular fold in the mucosa of the cervix becomes swollen and enlarged, and pushes its way backward through the original os uteri externum, which now constitutes a paler-colored ring about the base of the vaginal portion of the cervix. The os uteri externum of the cow is consequently a different structure from that of the heifer. Frequently there is a considerable infection in the first mucous fold, which causes it to be enormously enlarged, and its margins to become greatly enlarged and project out into the vagina as a series of pedunculated tumors. The entire muscular wall of the cervix becomes more or less thickened, as a consequence of the irritation within its canal, sometimes attaining a transverse diameter of four or five times the diameter in a normal heifer. In order to examine the cervical canal of the cow, the first annular fold of the mucosa is seized with the uterine forceps and the cervix is drawn well back into the vulva, where it can be inspected visually. If the forceps are drawn apart, any inflammation of the cervical mucosa can usually be quite clearly seen in the lips of the second annular fold. These are frequently scarlet or livid in color, showing an intense degree of inflammation, and in many cases there is a small amount of pus lying upon the mucous surface. Sometimes the principal inflammation is not in the posterior part of the canal, but in the anterior. In such cases, when the veterinarian introduces the uterine catheter to the anterior end of the cervical canal and causes some fluid to flow through it the first substance to escape is frequently a few drops of thick muco-pus, indicating that the cervical canal is severely inflamed at its anterior end —that is, at the posterior end of the uterus.

Cervicitis is one of the most dangerous types of inflammation

to be found in the genital tract. The infection tends very strongly to prevent conception, destroying the spermatozoa. If conception occurs, the infection lies in wait and presents two formidable dangers. It prevents the physiologic sealing of the cervical canal (the formation of the uterine seal) and is a permanent menace to the embryo, or fetus. In pregnancy, the infection tends to grow forward, radiating toward the apices of the horns from the cervical canal, causing a metritis which may finally bring about abortion, premature birth, or, if pregnancy continues to the full period, severe metritis, either with or without retained afterbirth.

When cervicitis is present, the cow should on no account be bred until the disease is under control. If the canal is very much narrowed and abruptly bent, it should be dilated, and in the process of dilating it is naturally straightened. When this has been done, the canal should be disinfected by swabbing with pure Lugol's solution or some other disinfectant of similar strength. It will withstand a most powerful disinfectant. In fact, it appears that a powerful disinfectant is necessary in order to bring about the required result. For swabbing, the cervix should be drawn well back into the vulva, the lips of the vulva held well apart with the forceps, a large dam of surgeon's cotton packed beneath the opening of the cervix, and a small swab of cotton, grasped with the uterine forceps and saturated with Lugol's solution, introduced along the entire length of the cervical canal. The treatment should be repeated about once in five to ten days, until all traces of cervicitis disappear, after which the animal may be bred.

It is to be borne in mind that cervicitis can not be cured so long as metritis exists, because while the latter continues pus flows constantly from the uterine cavity through the cervical canal and reinfects its mucosa. Consequently, in the handling of cervicitis, any metritis present must always be given proper attention.

It has been stated that in any scheme for the control of contagious abortion ample provision should be made for caring for the new-born calf. We have stated that the infection which exists in the pregnant uterus pursues a split course when pregnancy terminates: a portion of the infection remains in the mother, and another portion is expelled within the fetus. So far as known at present, the infection which is expelled with the fetus is generally

not in the fetal tissues and organs, but is stored up in the meconium or in some portion of the alimentary canal, where it appears to be held in leash until after birth has occurred. In some cases however, the infection is already in the tissues of the calf, so that it is ill at birth, and perhaps very quickly dies from septicaemia. In far more cases, the infection is lying more or less dormant in the alimentary canal, and within twelve to forty-eight hours after the birth of the calf reveals itself in the form of calf scours.

After birth, the calf may obtain the same infection from the mother in the act of nursing or by being fed upon her milk. The infection remains in the cow in two locations. The greatest and most dangerous volume is that which is left behind in the uterus. This infection largely escapes in the uterine discharges and flows down along the tail, buttocks, and udder, to contaminate the exterior of the udder and teats. From these, the calf swallows the infection with its milk, whether it nurses or the milk is drawn carelessly by a milker. It is now quite well established that the infection of contagious abortion — at least the Bang abortion organism — enters the udder through the teat canal, and persists for a long time in the gland. Then it escapes in the milk, so that the calf gets organisms which may cause calf scours or calf pneumonia, both from the exterior and the interior of the udder.

The third source of infection to the calf is from other calves which may be suffering from scours or pneumonia, either directly — by being placed in the same stall with the diseased calves, sucking them, picking up bedding which has been soiled by the excreta from diseased calves — or indirectly from contaminated feeding pails.

A fourth possible avenue of contamination is through the stump of the umbilic cord. Formerly this was considered the chief avenue of infection, but it must be relegated to an unimportant place, though it must still be regarded as having distinct dangers.

As at every other point in the handling of this chronic infection, so, in relation to calf scours and pneumonia, by far the most important element is the prevention of the infection. It is not known that the infection can be completely prevented in any

animal. So far as we know, those infections which cause calf scours and pneumonia are universally present in the adult, but the older animal has acquired a degree of resistance which prevents the infection from developing recognizable disease. The body of the new-born calf does not possess this power of resistance, and the volume and virulence of the infection are so great that it may prove a serious menace to the health or life of the calf.

First of all, if a virulent infection of the calf is to be prevented, it is essential that the uterus of the mother shall have been clean at the beginning of her pregnancy. Consequently it is important for the health and life of the calf, as well as for the breeding powers of the cow, that the uterine cavity, if seriously infected, should be disinfected prior to her becoming pregnant. In this way — and in this way only, so far as known — can we influence favorably the degree of infection which is to exist in the calf at the time of birth. If the calf is to be protected as far as possible against the infection existing within its alimentary tract when born, it should, first of all, be removed immediately from the cow, so that it shall not be permitted to suck. If the calf is allowed to suck, it takes into its alimentary tract a considerable volume of milk, which constitutes one of the best media in which the bacteria present may multiply. The calf does not need milk at all for twenty-four hours, or more, after birth. Hence, unless one is sure the calf is free from infection, milk should not be allowed. Instead, it is highly desirable that the expulsion of the meconium — the fetal feces — should be brought about by means of high enemas of salt solution, or by the use of purgatives. At present we are depending chiefly upon the high enemas administered by means of an ordinary hospital irrigator, with a pure gum horse catheter attached. If infection of the alimentary tract of the calf is suspected, or if the mother has retained afterbirth, which is virtually proof of such infection, important additional safety of the calf may be insured generally by the prompt administration of calf scours serum.

After twenty-four hours have elapsed, if the calf appears well, it may be fed about two per cent. of its body weight — that is, one pound of milk, twice daily, to a calf weighing fifty pounds. The calf scours serum may be continued once or twice daily until the

calf is three or four days old, when, if it appears well, the serum may safely be omitted. The milk ration may be increased gradually, day by day, until the desired amount has been reached.

The prevention of the infection of the calf through the milk of its mother is to be avoided chiefly by thorough bathing with an antiseptic of the entire posterior parts of the cow — especially of the udder — and drawing the milk from the udder in a sterile pail, under antiseptic precautions. When the infection exists within the cow's udder, the danger therefrom can not be avoided except by boiling the milk, which some new-born calves do not stand very well, or by procuring milk from a presumably healthy cow. When the calf is eight to ten days old, if it is well, the milk may be boiled or pasteurized with impunity, and if the calf is to be fed upon mixed milk the pasteurizing or boiling should be carefully carried out in all cases.

The third chief source of infection — contact with sick calves — can be avoided only by placing each new-born animal in a separate stall, with closed partitions, and keeping it in complete isolation until it is known to be sound, when it may be placed with other sound calves.

If calf scours sets in, the best remedy known at present is the calf scours serum, which may be given in almost any desired quantity, without apparent danger to the calf. Like other remedies, it can not always cure. If the calf is too thoroughly saturated with a highly virulent infection, it must die, but in a very large proportion of cases the calf may be saved. In addition to the liberal administration of calf scours serum, all food should be completely withdrawn for at least twenty-four hours, and enemas should be given to wash as much of the infection as possible from the bowel. A small dose of castor oil may be given to aid in cleansing the alimentary tract of the infection. After this has been done and the calf has had a liberal amount of serum, a small quantity of milk — one or two per cent. of its body weight — may be given twice daily. The feeding should be done very cautiously; the milk should be withheld if it does not seem to be digested and the scours continues. It should always be understood that the infection existing in the alimentary tract multiplies in the food given. Anything which will act as a food for the calf

will act also as food for the infection present in the alimentary tract, causing the scours. When food is placed in such an infected tract, the bacteria immediately swarm in it, producing poisons which are absorbed by the calf, intensifying the illness. On the other hand, the food, be it milk or other aliment, is not digested, and affords no nutriment whatever to the young animal.

The administration of the serum and other measures recommended should not cease as soon as the definite scours has ceased, but should be continued until the calf has recovered its vigor and strength. Calf scours serum should not be given with a view to saving the calf alone but also to controlling the infection and not permitting it to become thoroughly established in important volume in any part of the body. There is now such good evidence that the abortion and sterility so frequently seen in heifers is due to the infection which they acquired as young calves that the breeder is fully justified in taking every possible precaution against such infection, in order to insure the efficiency of the animal when it arrives at breeding age. Therefore, the object in the prevention and treatment of calf scours is a double one — the saving of the animal's life and the insuring of its efficiency as a breeding animal when it has reached maturity.

The Biologic Therapeutics of Contagious Abortion. In the modern practice of medicine, the use of vaccines, sera, bacterins, and other types of biologic product has attained a somewhat spectacular place. The accomplishments, first realized in smallpox, have in some diseases been truly remarkable. The outstanding successes have been with the acute — *i. e.,* immunizing — diseases, such as smallpox, diphtheria, tetanus, etc.

Several plans for controlling contagious abortion with the aid of biologic products have been recommended:

(1) It was early attempted to control contagious abortion by means of abortion bacterins or vaccines. Artificial cultures of the Bang abortion organism were grown, the bacilli killed, and liquid containing the dead bacilli and their excretions injected subcutaneously into pregnant cows and heifers. At first, abortion bacterins were very popular, as is any remedy suggested by any investigator of standing. As asserted in the report for 1911–

1912, it has always appeared to us illogical to hope for marked benefit from this source. In no case where we have been able to follow this line of effort, has anything been accomplished. In our report for 1915–1916, we give the history of eighteen heifers and their female progeny. Abortion bacterins were administered to these in 1912, during their first pregnancy, and the immediate and remote results — or want of results — are graphically indicated in Chart I of that report. Not only did the bacterins fail to control abortion during the pregnancy in which they were given, but the remote results were equally discouraging. Not only did the group abort with unusual severity, but the infection has followed the cows unrelentingly. Technically, the group should have increased by 1916 to 49 females of breeding age — the original 18, 27 daughters (9 each for 1912, 1913, and 1914) and 4 granddaughters from the 9 daughters of 1912 — a gain of 172 per cent. Actually there were, in 1916, 16 females of breeding age, counting those living of the original group of 18 and their female progeny — a decrease in the size of the group amounting to 11 per cent. Bacterins or vaccines are still advocated by a few makers of the product. The plan, tested by any known rule of therapeutics, is unsound, and, judged by any reliable records of experimental use, has failed.

(2) It has been proposed to control abortion by injecting living Bang abortion bacilli into the veins or beneath the skin, using the injection *before* conception. But a little while ago, essentially the same group of investigators was claiming that they could reliably *induce* abortion by the same means, by administering the cultures *during* pregnancy. Errors in diagnosis have caused a number of *pregnant* cows, supposed to be *non-pregnant*, to receive large amounts of live cultures as a preventive, and the statistics indicate that its power to prevent abortion was just as great as if it had been used *before impregnation*. The hyperinfection has, according to recorded data, *caused* abortion, when given for that purpose, and *prevented* it when given to prevent.

Hyper-infection, as a controlling force, is contrary to our present knowledge of contagious abortion. It is a chronic disease. If the animal is already infected — which we hold is almost, if not always true — the effort actually *superinfects* and renders the

life and efficiency of the animal more precarious. If, as claimed by most persons, contagious abortion is not universal, then intense artificial infection prior to pregnancy invites universal infection.

(3) Our researches have shown that the bacterial flora existing in the intestinal tract of the new-born calf is identical with, or at least indistinguishable at present from the bacterial flora existing within the utero-chorionic space of the pregnant cow. In the calf the infection is inseparably bound up with calf septicaemia, scours and pneumonia; in the cow with metritis, which may end in abortion, premature birth, and retained afterbirth.

It has been satisfactorily shown in practice that the congenital infections of calves are favorably influenced by the use of calf scours serum. This fact raised the question of the possibility of such serum, when given to the pregnant cow, affecting favorably the infection existing within the utero-choronic space.

It was recognized from the first that the two cases are not parallel. In the new-born calf, the bacteria are in some cases partly in the circulation and in various tissues open to the direct influence of the serum. When in the intestinal tract, where it exists most commonly and in greatest volume, it can not be directly affected by the serum, so far as known, but does affect favorably the walls of the alimentary canal, erects a barrier therein against intoxication, stimulates the bactericidal powers of the digestive tract, represses bacterial decomposition of the aliment, and causes in a large measure the expulsion of the infection as a part of the fecal mass. The infection is in immediate contact with the highly active intestinal mucosa. When the infection is within the utero-chorionic space, it is in wholly different environment. The mucosa of the utero-chorionic space is the most thoroughly neutral and dormant surface known in the animal body, and is hermetically sealed against external influences by the uterine seal closing the cervical canal. The active areas of both uterus and chorion are included in the placenta. The epithelial surfaces of the inter-placental areas of the uterus and chorion, so far as known, neither excrete nor absorb. Drugs or biologic products administered to the animal can not, so far as known, enter the space. They can not pass the placental filter, to reach the chorion, but can only reach the capillaries of the uterine mucosa. The infection within the utero-chorionic

space is therefore largely immune against attack by any substance introduced into the maternal blood stream. Neither is it subject to surgical interference, as this would necessitate the invasion of the uterus, involving the destruction of the uterine seal and the expulsion of the uterine contents.

In order to test the possibility of any influence of such a serum upon the infection in this location, the co-operation of a leading manufacturer of biologic products was secured, and that of the owners of a large herd of highly pedigreed Jersey cattle in which the abortion infection has long been severe. The department is deeply indebted to the manufacturers of the serum for their generosity in furnishing the serum, without cost, although at heavy expense to the firm. We appreciate equally the courtesy of the owners of the Jersey herd in permitting us to carry out the experiments. It speaks much for the earnestness of breeders in their desire to find a remedy for this scourge and their readiness to co-operate to that end.

The serum used was obtained from horses. The bacteria used in preparing the serum included the colon and other organisms usually found in scours, with the addition of the Bang abortion organism.

Twenty-six heifers were selected for the experiment. The rate of observed abortions in heifers during first pregnancy, over a series of eight or ten years, had been approximately 33 per cent. and it could be estimated with reasonable assurance that about one-third of the group of heifers selected would abort if left alone.

Each of the heifers was given the amounts of serum indicated in Table I on November 11 and 28 and December 13, 1916, and on January 24, February 20, March 26, April 27, and July 28, 1917. The final results are unknown in Nos. 21, 22, 23, 25, and 26. Pregnancy has terminated in nineteen heifers, with seven abortions (36.8 per cent.), two premature births, and ten calves born at two hundred and sixty-nine to two hundred and eighty-four days. Of the seven abortions, three received forty-mil doses of serum, and four received twenty mils at each dose. Neither of the controls has thus far aborted, though one, No. 11, calved prematurely and had retained afterbirth.

Without awaiting the final result, it is quite evident that the serum used exerted no favorable influence. Thus three different

plans for using biologic products for controlling abortion — living Bang abortion bacilli (hyper-infection), dead Bang abortion bacilli (bacterins), and abortion serum — have been tried. So far as can now be seen, the opinion expressed by us, that biological treatment of abortion is necessarily fruitless, apparently holds true.

TABLE 1

NUMBER	Born	Dates of breeding	Amount of serum (mils)	Dates of Administration								Pregnancy ended	Duration in days	Result
				1916			1917							
				Nov. 11	Nov. 28	Dec. 13	Jan. 24	Feb. 20	Mar. 26	April 27	July 28			
1	11/ 8/14	3/29/16 4/19/16	40	+	.....	.....	.....	.....	.....	.....	.....	11/28/16	223	X
2	11/14/14	2/22/16 3/14/16	40	+	+	.....	.....	.....	.....	.....	.....	12/10/16	271	C
3	12/16/14	5/ 1/16	20	+	+	.....	.....	.....	.....	.....	.....	12/ 5/16	218	A
4	12/31/14	4/ 2/16	20	+	+	+	.....	.....	.....	.....	.....	12/28/16	270	C
5	11/25/15	12/ 2/16	Control	—	—	—	—	—	—	—	—	8/28/17	269	C
6	2/18/15	12/ 7/16 1/ 2/17 1/16/17 2/10/17 3/ 2/17 3/25/17 4/13/17	40	+	+	+	+	+	+	+	+	12/12/17	243	A
7	2/13/15	12/ 4/16	40	+	+	+	+	+	+	+	+	8/10/17	249	A
8	3/14/15	7/14/16 8/19/16	40	+	+	+	+	+	+	+	+	4/30/17	254	A
9	3/ 7/15	12/ 5/16	20	+	+	+	+	+	+	+	+	8/21/17	259	A
10	4/10/15	12/ 4/16 12/23/16 1/12/17 2/ 8/17 2/27/17 3/29/17 4/17/17 5/ 8/17	20	+	+	+	+	+	+	+	+	.....	.....	P 10/30/17

		6/ 5/17 6/27/17												
11	5/ 3/15	12/ 4/16	Control	—	—	—	—	—	—	—	—	7/27/17	220	X. R.
12	5/19/15	12/ 6/16 4/ 2/17 4/22/17 5/25/17 6/27/17 8/27/17	40	+	+	+	+	+	+	+	+	.....	.....	10/30/17 P. 50 to 60 ds
13	5/21/15	.....	40	+	+	+	+	+	+	+	+	4/ 6/17	.....	C
14	6/22/15	12/ 2/16	20	+	+	+	+	+	+	+	+	9/12/17	284	C
15	7/27/15	12/14/16 1/ 4/17 1/24/17	20	+	+	+	+	+	+	+	+	10/28/17	284	C
16	8/17/15	12/12/16 12/30/17 2/22/17	Control	.....	.....	.....	.....	.....	.....	.....	.....	11/20/17	271	C
17	8/19/15	12/13/16 1/23/17	40	+	+	+	+	+	+	+	+	11/ 2/17	283	C
18	9/ 1/15	11/30/16	40	+	+	+	+	+	+	+	+	8/28/17	271	C
19	9/13/15	12/ 2/16 1/ 7/16 1/29/16	20	+	+	+	+	+	+	+	+	11/ 3/17	278	C
20	10/19/15	Bred in pasture	20	+	+	+	+	+	+	+	+	2/28/17	?	A
21	11/12/15	6/ 1/17	Control	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	P
22	1/ 7/16	11/13/17	40	+	+	0	+	+	+	+	+	.....	.....	P?
23	1/17/16	11/17/17 12/14/17	40	+	+	+	+	+	+	+	+	.....	.....	?
24	1/22/16	Bred in pasture	20	+	+	+	+	+	+	+	+	10/19/17	.....	A
25	2/26/16	11/13/17	20	+	+	+	+	+	+	+	+	.....	.....	P?
26	5/14/16	11/26/17	Control	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	P?

Anaphylaxis, or Serum Disease.— While the phenomenon of anaphylaxis has been seen and partially understood by the medical profession for some years, especially since the introduction of diphtheria and tetanus antitoxin, little has been said about it in veterinary literature. Laboratory workers have produced the condition experimentally in all domestic animals. Bang, in one of his experiments on cattle, witnessed the reaction. Hadwen and Bruce,[1] and others, have produced anaphylactic shock in cattle by injecting intravenously extracts of crushed parasites (larvæ of Hypoderma bovis) into animals infested with the same species. The average practitioner is not apt to be confronted with the reaction from these sources, but rather, as in the human family, from the prophylactic or therapeutic use of biological products. Concerning the reaction from this source, we have seen or heard very little.

Anaphylaxis may be thought of as the development in an animal under certain conditions of a hypersusceptibility of the body cells to a foreign protein, which, in itself, is not ordinarily toxic. Under these circumstances, such ordinary proteins as milk, egg white, or blood serum may produce grave distress and even death, when injected parenterally in very small quantities.

When most antigenic substances are injected into the animal body, a reaction ensues which results in the production by the body cells of a substance (antibody) which makes the individual less reactive or susceptible to a following dose of the same substance. This is spoken of as immunity. It may be produced toward practically any protein substance — *e. g.*, bacterial bodies, bacterial toxins, serpent venoms, egg-white, milk, foreign blood serums, etc. Under other circumstances, which are as yet imperfectly understood, an entirely different reaction occurs. After the first injection, the body cells after a certain interval become more reactive or hypersensitive to a second injection. When the second injection is given, therefore, a violent reaction or anaphylactic shock may take place. From the fact that the first cases studied were those occurring through the use of serum in treating diphtheria, the phenomenon is commonly called "serum disease" or "serum sickness."

[1] Jour. Am. Vet. Med. Assn., Vol. II, New Series, Vol. IV., No. 1, Apr. 1917, p. 15.

The practitioner who uses any of the several serums or antitoxins in his practice is apt, sooner or later, to meet "serum disease." This is due to the fact that many of the commercial products are made, for economic or other reasons, from a different species of animal than the one on which the product is to be used. The serum which is injected contains a protein which is foreign to the body. If the serum is one of the same species, no trouble will ordinarily result, for the serum is then homologous — *i. e.,* the protein is not foreign.

For the production of anaphylaxis, a definite incubation period is necessary. How long this period is for our larger animals, we do not know. If the primary injection is followed after a few days by another, no reaction will ensue, but rather immunity develops. If the incubation period has elapsed before another dose is injected, anaphylactic shock is apt to occur. Several doses close together, following the primary dose, serve to lengthen the incubation period.

In the use of an antitoxin, or immune serum, prophylactically or therapeutically, we have two processes going on simultaneously, which should not be confused. If the injections are so spaced that anaphylaxis or hypersensitiveness ensues, it must not be thought that the animal is made more susceptible to the particular disease toward which we are attempting to immunize it. Such is not the case. The reaction is between the body cells and the particular protein of the serum which is present in a non-immune serum as well. The anti-bodies of the immune serum are carried over unaffected by the anaphylactic shock which occurs, and immunity results the same as if the reaction had not occurred.

In the experiments upon the heifers recorded in Table I, the serum had been injected on November 11 and 28, and December 13, 1916, and January 24, February 20, March 26, April 27, and July 28, 1917. No symptoms of anaphylaxis were noted following the administrations on November 28 or December 13, 1916. Between the injections of November 11 and 28, there had elapsed seventeen days; between November 28 and December 13, fifteen days; and between December 13, 1916, and January 24, 1917, forty-two days.

The administration of the serum on January 24, 1917, was

begun on the group at approximately 8:30 A. M. and completed at 9:30 A. M. We had been watching closely for anaphylaxis or other unusual symptoms following the injections upon the first three dates, but nothing had been observed, and it was assumed finally that neither anaphylaxis nor other untoward symptom was likely to develop. Accordingly, without detailed observations of the animals, as soon as the injections had been completed, we went to another stable, about two miles distant, and were engaged in other work. Within thirty minutes after we had left the stable, the man in charge of the heifers observed that the eyelids of some of them were swollen and that a profuse lacrimation was present. The symptoms continued to develop quite rapidly, and in a little while the caretaker observed that all, or essentially all of the heifers which had received the serum were involved. The keeper finally became quite alarmed and telephoned for us to return.

Returning to the stable, after an absence of two or three hours, we found the symptoms apparently abating. All the heifers were involved. While there were variations among the heifers, these did not seem to be caused by the amount of serum. Those which had received twenty mils each were apparently suffering as greatly as those which had received forty mils each. If there was any difference, those which had received the larger amounts were somewhat worse.

The eyelids were enormously swollen and edematous, as though they were suffering from the stings of bees. Tears were flowing abundantly over the cheek, so that a large area was wet and tears were dropping to the floor. The condition of the eyes gave the heifers a very dull aspect, but in spite of this nearly all of them were ruminating or feeding. As the heifers were very well fed, there was nothing remarkable about a few of them not eating or ruminating for a while. The vulva of each heifer was greatly swollen and edematous; the labiæ were at least twice their normal volume and the skin over them deeply injected and glistening. The udder also was greatly swollen, red, and glistening. Several of the heifers were trembling violently, as if suffering from a severe chill. Two or three of them were sweating profusely. One of the heifers breathed stertorously: she could be heard readily at a distance of 100 feet. Five hours after the serum had been given,

one heifer continued to sweat and tremble, and another to tremble. The vulvae and udders remained about the same in all. The edema of the eyelids and the flow of tears diminished rapidly. Nearly all the heifers were either feeding or ruminating. Twenty-four hours after the giving of the serum, the heifers were essentially normal, except that the vulvae and udders remained somewhat swollen and the edema of the eyelids was still recognizable.

Slight symptoms of serum disease were observed upon three animals following the administration of serum on April twenty-seventh. Following the later administrations of the serum, no symptoms of serum disease developed.

While the symptoms were for a time quite severe and tended to cause alarm, apparently no harm ensued, and the heifers quickly became normal. It seems peculiar that the serum disease should have developed at this particular time instead of following the administration of the second dose. Apparently the second and third doses followed too closely upon each other to permit of sensitization, and it was only after the interval of forty-two days between the third and fourth injections that the sensitization to the serum became developed. How much or how little danger there may be from serum disease in cattle when the horse is used for producing the serum, is not brought out by this observation. Apparently in the ordinary use of calf scours serum upon calves, the doses follow each other so rapidly that no sensitization occurs, and consequently no anaphylaxis. The only practical question, so far as can be seen, which may arise from these observations is the possibility, when the horse is used for the production of calf scours serum, that if the serum is used upon a new-born calf and then omitted for several weeks, and used again, it might possibly possess danger. In our experiment however, with the twenty-one animals involved, no serious consequences developed, and there is no definite evidence to show that there is any very great danger of producing serum disease in the administration, where all the serum used is given within the first five or ten days of the calf's life.

GENITAL TUBERCULOSIS OF CATTLE

Tuberculosis of the genitalia of cattle has generally been considered as rare and of scant scientific and economic importance. William Williams[1] states, "If the cow be in calf, abortion is apt to occur; if not pregnant, the condition called nymphomania is frequently present." Law[2] states, "The generative organs also occasionally suffer, in which case an early and rather persistent symptom is sterility, with a too frequent, or it may be persistent desire for the bull (nymphomania). * * * In cases of uterine tuberculosis, the nymphomania may be supplemented by a purulent discharge. * * * Genital tuberculosis in the bull is associated with nodular swelling of the testicle, epididymis, or cord, hydrocele, and exceptionally tubercle on the penis or in the prostatic sac." Friedberger und Fröhner[3] state that genital tuberculosis occurs rarely in both sexes and may invade any portion of the genital system. Hutyra und Marek[4] mention tuberculous epididymitis and orchitis, and assert that rarely the testis undergoes tuberculous abscessation, with a consequent fistula. They consider penial tuberculosis as extremely rare, and claim that when it occurs the glans penis becomes studded over with tubercles. Quoting Hess, they regard uterine and tubal tuberculosis as rare and as causing sterility and nymphomania. Vulvo-vaginal tuberculosis is mentioned as a rare possibility. Hoare[5] describes genital tuberculosis very briefly, without according it an important place. He admits the possibility of transmission by copulation, and mentions a single case of primary penial tuberculosis.

The general trend of veterinary literature concerning genital tuberculosis is in sharp contrast with my personal observations. The difference in view is possibly due to a variation in the direction of study. Veterinarians rarely look for genital tuberculosis, since ordinarily it does not affect the general well-being of the patient. Clinically, it is only when one concentrates his observations upon the diseases of the genital organs in connection with

[1] Principles and Practice of Veterinary Medicine, 1875, p. 347.
[2] Vet. Med., Vol. IV, 1902, pp. 448, 449.
[3] Speciellen Pathologie und Therapie.
[4] Spezielle Pathologie und Therapie der Haustiere.
[5] System of Veterinary Medicine.

sterility that the recognition of genital tuberculosis becomes probable and its importance realized. The genitalia are not inspected frequently in the abattoir. They are not used as human food. When they are tuberculous, the lesions possess little or no importance in relation to the value of other tissues for food. Hence, much genital tuberculosis may pass unnoted.

The genital mucosa offers a highly vulnerable field for tuberculous invasion, but genital exposure to tuberculosis is rare as compared with exposure through contaminated food. That is, when open pulmonary tuberculosis exists, the patient is constantly contaminating mangers, food, and water by means of her sputum, so that companions are exposed daily and hourly. When genital tuberculosis exists, the exposure may be identical, because of the genital discharges contaminating food and water, but the special venereal exposure occurs only during the very brief period of copulation.

I have not observed tuberculous orchitis or epididymitis. There is no example of either in my pathologic collection of genitalia, and no specimen in the collection of any department of the college. The scattered records in veterinary literature of tuberculous orchitis and epididymitis do not serve as a very accurate basis for outlining the clinical symptoms. In a general way, it is stated that the epididymis and testes show painless enlargement and hardening. The demarcation between the epididymis and the testis gradually becomes clouded, and finally lost. Apparently the epididymis usually becomes involved first and the disease extends thence to the gland. The location of the tuberculous process is said to be usually in the parenchyma, having its basis in the mucosa of the epididymis and testis. In this manner the disease may be well advanced before peripheral inflammation or tuberculous extension involves the peritoneal coverings, to cause adhesions and hydrocele. Rarely, abscessation is said to ensue, resulting in a fistula.

The clinical diagnosis of tuberculous epididymitis and orchitis is difficult. The painless tumefaction of the testis is not characteristic of tuberculosis, but may ensue from various pyogenic infections. The peritoneal adhesions, hydrocele, and abscessation are quite as probable from other infections as from tuberculosis.

Tuberculin offers, next to the excision of the testis and its examination, the best means for diagnosis. As is well known, tuberculin has its limitations, and may fail. If the patient responds to tuberculin, the evidence of tuberculous epididymitis or orchitis is not complete. Tuberculous lesions in other organs may cause the response to tuberculin, while the lesions in the epididymis and testis may be non-tuberculous. When but one testicle is involved, its removal and histo-biologic examination offers by far the most reliable means for diagnosis. If the disease has existed for some time, the removal of the testis is in no case of economic importance, because as a general rule chronic epididymitis or orchitis signifies incurable sterility of the involved gland. Hence its removal is no loss, but actually, as a rule, the best curative measure and the greatest available protection for the other testicle and for the breeding life of the bull.

The path of tuberculous invasion of the epididymis and testis has not been clearly learned. It is not primary. It does not seem to be secondary to penial infection. While I have observed several cases of primary penial tuberculosis, I have not noted subsequent tuberculous epididymitis or orchitis. It appears highly improbable that the tubercle bacilli would traverse the long urethra and vas deferens and reach the epididymis without leaving behind evidences of its passage in the form of penial or urethral tuberculosis. When wholly within the tubules of the epididymis or testis, as appears usually to be the case, the invasion is apparently not direct from the peritoneum. When the scrotal peritoneum is first involved, it would appear probable that the invasion had occurred from the peritoneal cavity through the open inguinal ring. Apparently most instances of tuberculous epididymitis and orchitis are referable to hematogenic sources and constitute a part of generalized tuberculosis.

The prognosis for the involved gland is hopeless. If only one gland is involved, it may be successfully removed, leaving the bull perfectly fertile. The difficulty is that the tuberculous epididymitis or orchitis is usually a secondary rather than a primary lesion and the basic lesion remains and probably unfits the bull for breeding except when mated with infected cows held under the Bang plan. Even in such cases, it needs be determined that the

vas deferens, vesicula seminales, prostate gland, and penis are free, or the bull becomes a serious menace even to tuberculous cows, because when such parts are involved tubercle bacilli may be ejaculated with the semen from the sound testicle and induce primary genital (venereal) tuberculosis in the cow, which may promptly bring her breeding life to a close.

Tuberculosis of the vas deferens, seminal vesicle, and prostate is possible, but apparently too rare to be of great economic importance. Tuberculosis of the vesicula seminales or prostate would tend to interfere with urination. The diagnosis of tuberculosis of these parts would be based necessarily upon rectal palpation.

Penial Tuberculosis.— Penial tuberculosis is comparatively common in the bull. The corpus cavenosum, urethra, and urethral mucosa are not so frequently involved as the submucosa of the glans, prepuce, sheath and the adjacent penial lymphatics.

Tuberculosis invades any organ far more readily when the protective epithelium is wounded. The anatomy of the copulatory apparatus of the bull and the mechanism of copulation render epithelial injuries more probable elsewhere than in the mucosa of the glans penis itself. The penis of the bull (and of ruminants generally) is very firm. The transverse diameter and the consistency of the penis are but slightly modified by erection. The protrusion of the penis during copulation is brought about almost wholly by the elimination of the sigmoid flexure (See Fig. 12), the accomplishment of which requires the relaxation or overcoming of the retractor penis muscle. When at rest, the penis lies within the prepuce, above the sheath. The mucosa of the sheath of the adult bull is not commonly distinguished from that of the prepuce, although they have a wholly different embryologic history. The sheath exists as a distinct structure at the time of birth; the prepuce does not. In the new-born ruminant and porcine male, the sheath constitutes a comparatively short infundibulum, terminating at the bottom in the meatus urinarius (Fig. 1). No part of the glans penis is exposed. At the time of birth, the two layers of mucosa, one of which is later to form the mucosa of the glans penis and the other the mucous membrane of the prepuce, are firmly adherent to each other, as shown in Figures 1 and 2. Later, when sexual maturity approaches, the tissue

between the two layers of mucous membrane which serves to bind them together slowly gives way, and the perputial sac finally becomes established. In the abattoir, one may observe every gradation of the development of the prepuce in veal calves. The gradations may be seen also in castrated males (Fig. 3). The prepuce may be totally absent, or there may be any degree of development, according to the degree of sexual development at the time of castration. The prepuce of the bull therefore represents that portion of the penial envelope extending from the apex of the glans penis to the base when the penis is at rest. The sheath consists of that portion extending from the apex of the glans penis forward to the sheath opening.

The new-born ruminant or porcine male can not protrude the penis, and if castrated early never becomes able to do so. The embryonic history is traceable in the anatomic characters of the two membranes. The preputial epithelium is very delicate and easily abraded; the epithelium of the sheath is coarser and offers higher resistance. The preputial mucosa is even and smooth; the sheath mucosa is thrown into wavy longitudinal rugae.

In coitus, the protrusion of the penis is effected by momentary elimination of the sigmoid flexure (Fig. 12) which necessitates the elongation of the retractor muscle of the penis. When the glans is protruded beyond the prepuce and sheath, the prepuce and the sheath each becomes reflected upon the penis, to constitute its outer covering. For the moment, both preputial and sheath cavities cease to exists, and the mucosa of each, instead of lining its respective sac, constitutes the external covering of the penis. The reflection of these two membranes is made possible by the very loose areolar connective tissue lying between the mucous lining of the canal and the outer yellow elastic framework of the sheath derived from the abdominal tunic. The coital thrust of the bull is vigorous, or even violent. It is so violent that occasionally, as shown in Fig. 8, the prepuce is torn or ruptured. The abrasion or rupture is withdrawn at once, with the retraction of the penis, and there is carried along any infection which has entered the wound.

The superficial genital mucosa of the bull, as well as that of the ram and the boar, is further exposed to abrasions and consequent

infection by the presence of the granules or nodules of the granular venereal disease — a chronic infection which is essentially universal. The granules or nodules frequently become inflamed, especially from excessive coitus, the epithelium at the summits of the granules becomes abraded, and hemorrhage ensues. It is not rare to see the granular venereal disease so severe that the parts bleed after each coitus, if the bull will copulate. Sometimes the pain is so great that he refuses to copulate; sometimes the swelling of the sheath is so great that the penis can not be protruded — the bull has phimosis.

Therefore, in addition to the very delicate epithelium of the genital mucosa, the prepuce itself may be ruptured, or there may occur a multitude of minute abrasions at the summits of the granules regularly present. These abrasions offer special facility for invasion by pathogenic bacteria. If the cow is afflicted with tuberculous genital catarrh, any abrasions upon the penis, prepuce, or sheath of the bull existing or occurring at the time of copulation invites tuberculous infection. The entire group of such tuberculous infections may be classed as primary venereal or coital tuberculosis. Primary tuberculosis of the male copulatory organs of the bull may involve any tissue or part contributing to the copulatory apparatus. The principal types I have observed are:

(1) Tuberculosis of the glans penis.
(2) Sheath tuberculosis.
(3) Preputial tuberculosis.
(4) Tuberculosis of the penial lymph glands.

(1) *Tuberculosis of the glans penis* is not very rare. I have observed two cases clinically, and others in the abattoir. One clinical case was in a large Holstein herd bull. For some months, he had been unable to copulate with small or medium-sized cows, but could do so successfully with large cows with commodious vulvae. Attempts at copulation were generally followed by a limited amount of hemorrhage. The glans penis was enlarged, especially at its apex. The surface was dark-colored, congested, somewhat eroded, and suppurating slightly. Inability to copulate was apparently referable to the combination of four factors — the enlargement of the glans; the roughening of its surface,

due chiefly to the destruction of the epithelium, thus hindering the introduction of the glans into the vulva; the pain; and the flaccidity of the diseased area. The penis was protruded readily. When the bull was confined upon the operating table, the penis could be pushed out of the sheath and prepuce by forcibly effacing the sigmoid flexure. When thus forced out and securely grasped so that the part could be readily inspected, it was seen that the apex of the glans for a distance of about 3 inches was much inflamed and enlarged, and bled readily upon touch. The diseased tip was dark livid, the epithelium largely destroyed, and the surface contaminated by purulent exudate. The appearances were strongly suggestive of tuberculosis. The diseased tip of the glans was amputated. The histologic appearances were those of tuberculous lesions, and stained smears showed tubercle bacilli. The operative wound progressed very favorably for a time, and the bull was promptly discharged from the clinic, apparently on the safe road to recovery.

The patient belonged to a breeder devoid of serious regard for the control of tuberculosis. It was understood that the disease was rampant in his herd and that he was more concerned about concealment than about control. I was unable to follow the case. Apparently the patient, valued at about ten thousand dollars, had contracted the infection by copulating with a cow having genital tuberculosis. After becoming infected, he was evidently a very serious menace to any cow free from genital tuberculosis with which he might copulate. The venereal peril was limited chiefly by the fact already related, that he could not copulate with most cows and heifers, but only with those having commodious vulvae. The attitude of the owner prevented any investigation of the herd in an effort to learn whether any harm had come to cows from copulation with this animal. So far as I have observed, he was one of the most dangerous bulls I have seen with genital tuberculosis, because in most cases copulation is more promptly and absolutely excluded and venereal transmission thereby avoided.

A very interesting abattoir specimen is illustrated in Fig. 8, in which there has been a rupture of the prepuce and also tubercle infection at the tip of the glans penis. I see no relation between the two lesions.

Tuberculosis of the submucosa of the glans is illustrated in Fig. 5. This specimen shows numerous tubercules in the submucosa. They were not recognizable clinically. The disease was brought to a crisis by the larger tuberculous abscesses in the prepuce and about the base of the glans, which rendered protrusion of the penis impossible.

(2) *Tuberculosis of the penial sheath* is presumably rare. I have but two well defined specimens in my collection. Both were obtained from the abattoir, and are without clinical history. One, Fig. 4, indicates that penial incarceration occurred early, rendering the animal impotent and causing him to be sent to the shambles. The other, Fig. 10, is a more complex case with rupture of the prepuce and tuberculosis of both glans and sheath. Clinically, the condition could not well have been differentiated, upon ordinary examination, from other infections of the sheath wall, especially actinomycosis. Amongst chronic infections however, tuberculosis is by far the most probable, and a provisional diagnosis of tuberculosis should be made. The tuberculin test may serve as an aid, but is subject to severe limitations, since any reaction may be due to pre-existing tuberculous lesions in other parts of the body. If it is reasonably certain that the bull was tubercle-free prior to the development of the lesion, the test possesses great value. Exploratory incision through the external skin, and the removal of material for bacterial and inoculation studies, is of great diagnostic value.

So long as the bull can protrude the penis and copulate, he constitutes a serious menace. Since the infection is primary, the fundamental lesion is in the mucosa, and naturally open, and any tuberculous excretions occur within the sheath. The glans penis inevitably becomes contaminated, but not necessarily infected. In copulation, however, the surface contamination is carried into the vulva and vagina of the female. In protruding the penis also, as the reader will understand by studying Figures 6 and 7, until the swelling becomes too great for copulation to occur, the sheath mucosa is reflected and when the penis is fully protruded the tuberculous membrane itself constitutes the covering of the penial base and enters the vulva of the cow during coitus.

Accordingly it is essential that great care be exercised in handling inflammatory conditions of the sheath and, in making a diagnosis, that every available means be taken to eliminate the question of tuberculous infection. However, one must not incise the sheath deeply, for diagnostic or other reasons, lest the resultant cicatrix incarcerate the penis. If tuberculosis is diagnosed, the bull should be excluded from service, even to reacting cows, and promptly sent to slaughter.

(3) *Preputial tuberculosis* is apparently less common than tuberculosis of the glans or of the sheath. The preputial membrane, which is far more delicate than that of the sheath, is exposed during copulation to special injury at the base of the glans. At this point the epithelium of the glans becomes reflected, to constitute the lining epithelium of the preputial sac. At the moment of the copulatory thrust, the parietal, or outer preputial membrane is suddenly and violently reversed, to constitute the covering of the penis from the base of the glans backwards for a distance approximately equal to the length of the glans itself. Further back, the sheath mucous membrane succeeds the prepuce as the mucous covering of the protruded penial body. The prepuce and the sheath mucosa become completely reversed. That end of the sheath mucosa which, while the penis was at rest, was situated most anteriorly now becomes most posterior. The mucosa of the sheath and of the prepuce become reversed in relation to each other. When the penis is at rest, the prepuce is behind the sheath, but when the penis is protruded the prepuce is in front of the reversed sheath mucosa. Accordingly the prepuce, at the point of its attachment to the base of the glans, is one of the most vulnerable points at which tuberculosis and other infections may effect an entrance. When infection occurs, it usually leaves scant trace in the epithelium of the glans, but involves chiefly the lymph glands of the submucosa.

As soon as infection occurs and inflammation is established, the loose areolar tissue between the prepuce and the external dartoid sheath becomes involved, adhesions occur, and the penis is incarcerated. By studying Figures 1, 2, 6, 7, 9, and 10, it will be seen that as soon as such inflammatory adhesions become established copulation is at an end, because the preputial wall can not

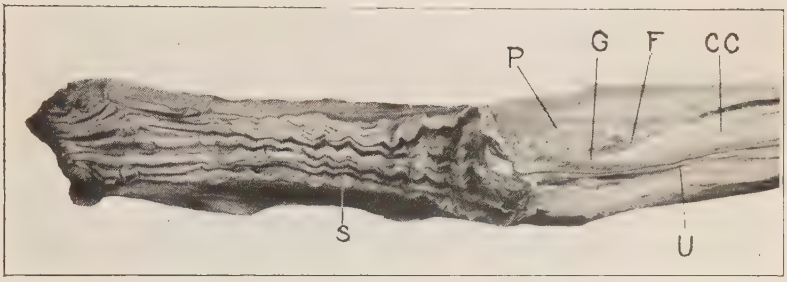

FIG. 1.— PENIS AND SHEATH OF BULL CALF, IN SAGITTAL SECTION.
S, sheath; U, urethra; P, prepuce, not yet free from the glans, G, the interval being occupied by soft embryonic tissue; F. fornix; CC, corpus cavernosum.

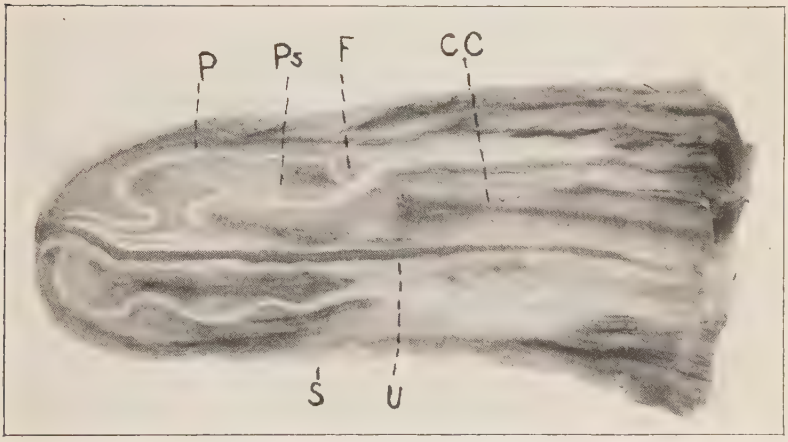

FIG. 2.— PENIS OF BULL CALF WITH THE SHEATH FORCIBLY REFLECTED OVER THE GLANS.
Ps, preputial sac, occupied by embryonic tissue. Other letters same as in Fig. 1.

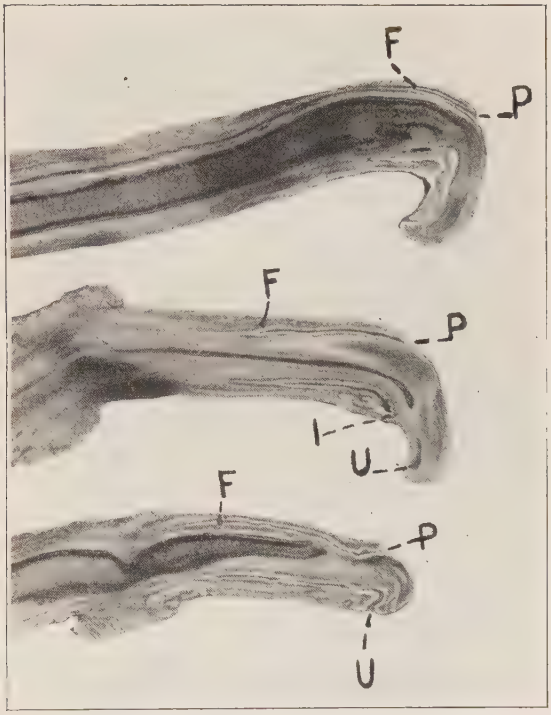

Fig. 3.—Sagittal Sections of Penises of Steers, Showing Gradations in the Dehiscence of the Prepuce from the Glans.

P, prepuce; F, fornix; U, urethra; 1, adhesive band on median raphe.

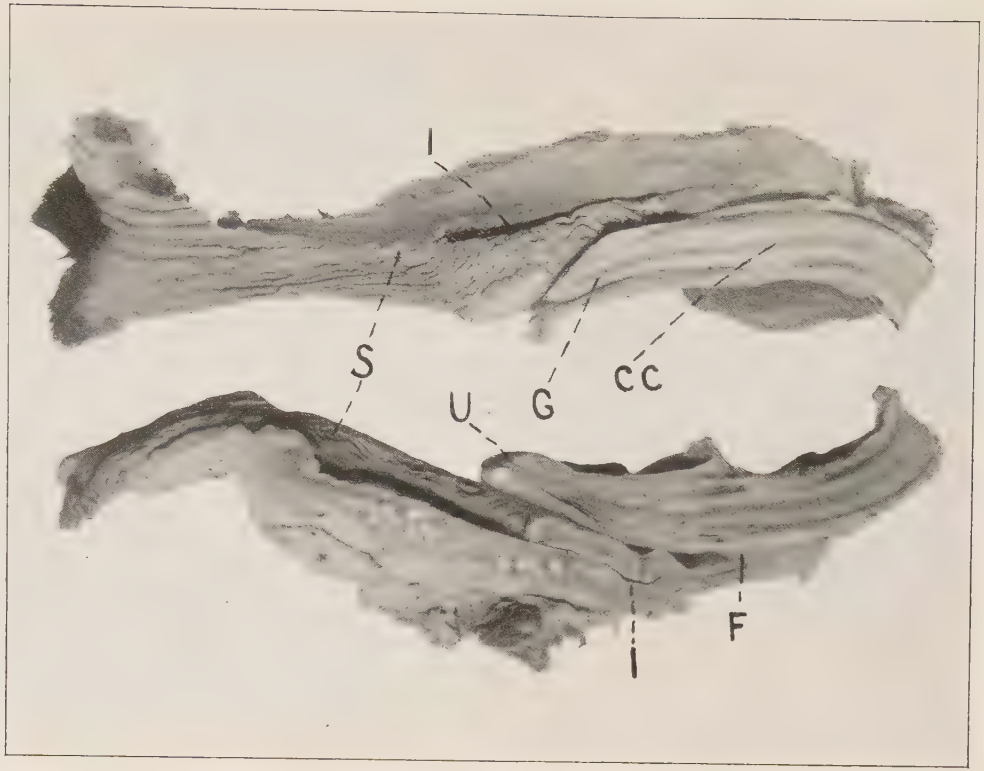

FIG. 4.— TUBERCULOSIS OF SHEATH AND PREPUCE OF BULL.
S, sheath; U, urethra; G, glans; F, fornix; CC, corpus cavernosum, 1,1, fissure extending into tubercular mass.

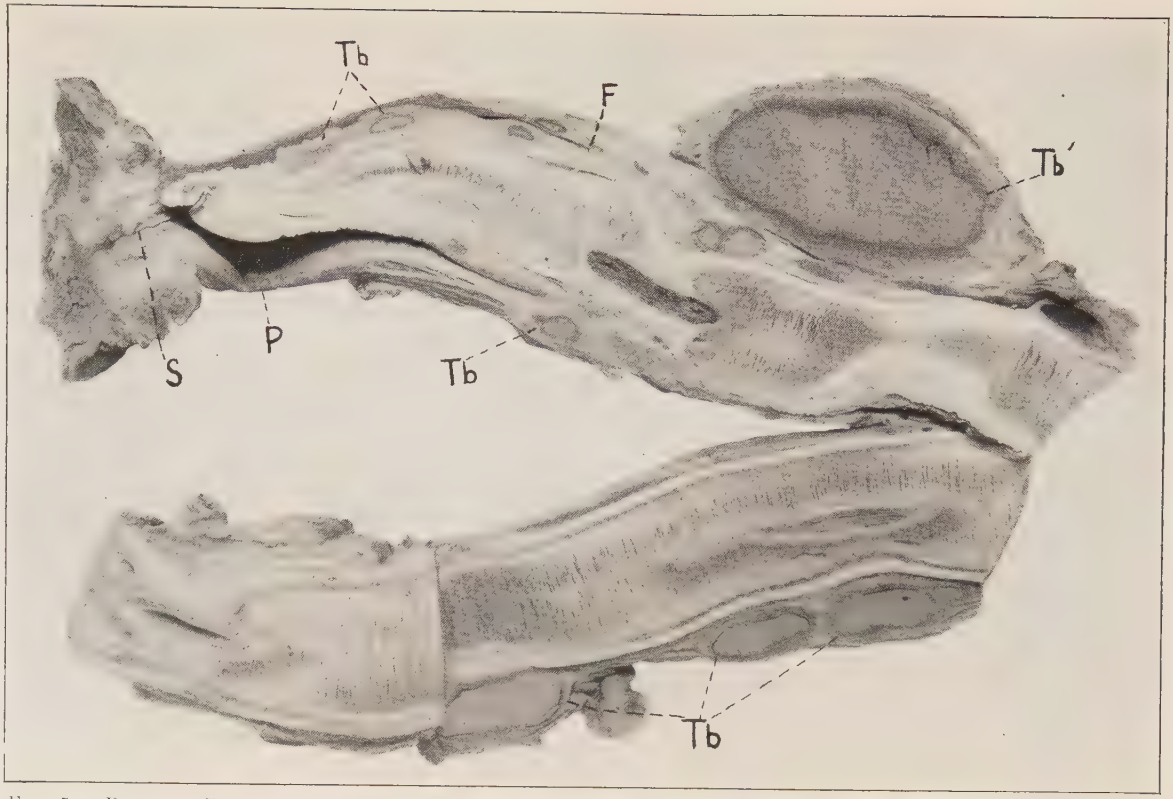

FIG. 5.— PRIMARY PENIAL TUBERCULOSIS IN BULL. SAGITTAL SECTION, WITH CROSS SECTION AND DOUBLING. S, sheath; P, prepuce (ventral side); Tb, tubercular masses; Tb', large tubercular abscess at base of glans. The dotted lines from Tb, at the left of the fornix, F, indicate tubercular masses in the submucosa of the glans.

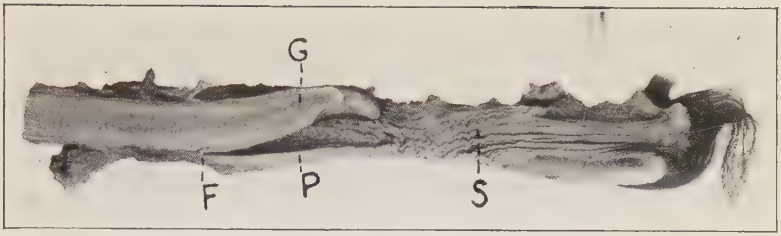

FIG. 6.— SAGITTAL SECTION OF PENIS OF BULL, RETRACTED WITHIN THE PREPUCE.

F, fornix; G, glans; P, prepuce; S, sheath.

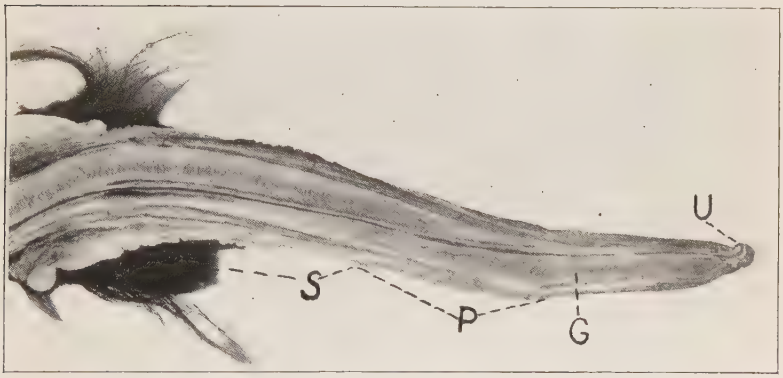

FIG. 7.— PENIS OF BULL, PROTRUDED FROM SHEATH AND PREPUCE.

Lettering same as in Fig. 6.

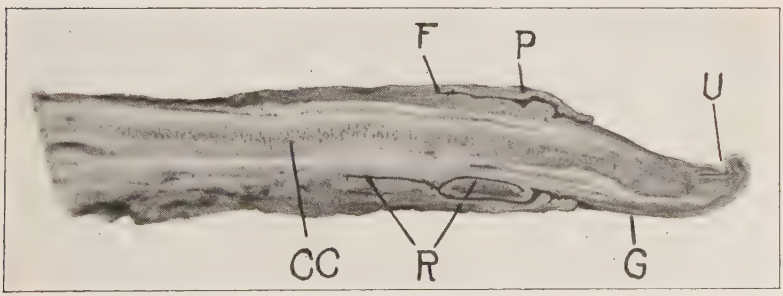

FIG. 8.— RUPTURE OF PREPUCE FROM COITAL VIOLENCE.

CC, corpus cavernesum; F, ferrix; P, prepuce; G, glans; U, urethra; R, coital rupture. At the right end of the rupture the ruptured border of the prepuce is folded upon itself.

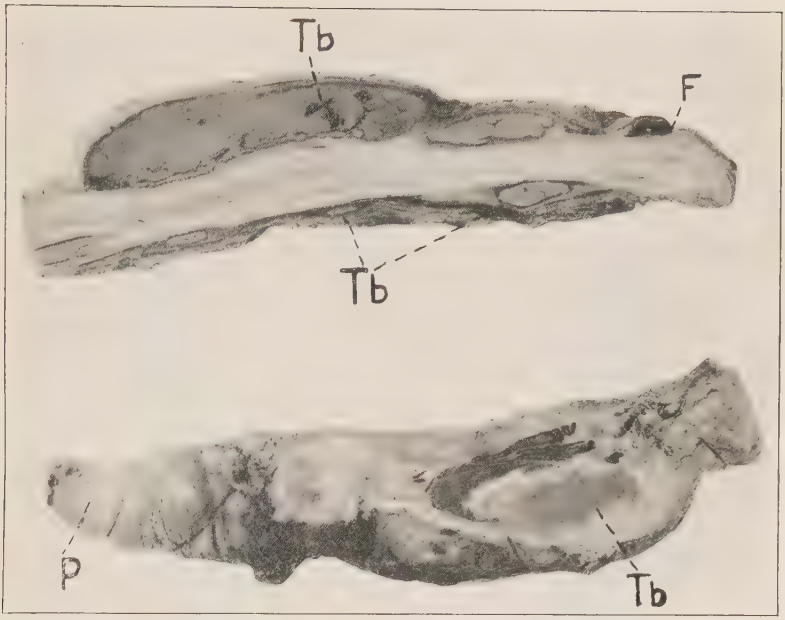

Fig. 9.— Tuberculosis of the Glans and Penis.
P, prepuce; F, fornix; Tb, tubercular masses.

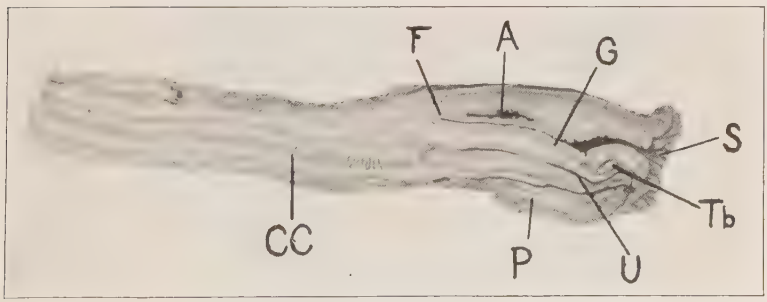

Fig. 10.— Tuberculosis of Glans and Prepuce.
S, sheath; G, glans; P, prepuce; F, fornix; U, urethra; CC, corpus cavernosum; Tb, tubercle in tip of glans; A, tubercular abscess in prepuce.

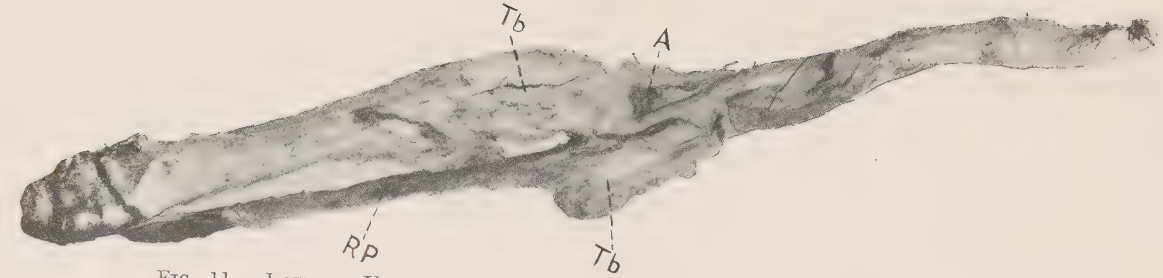

FIG. 11.— LATERAL VIEW OF PENIS OF BULL WITH TUBERCULAR PENIAL ADENITIS.

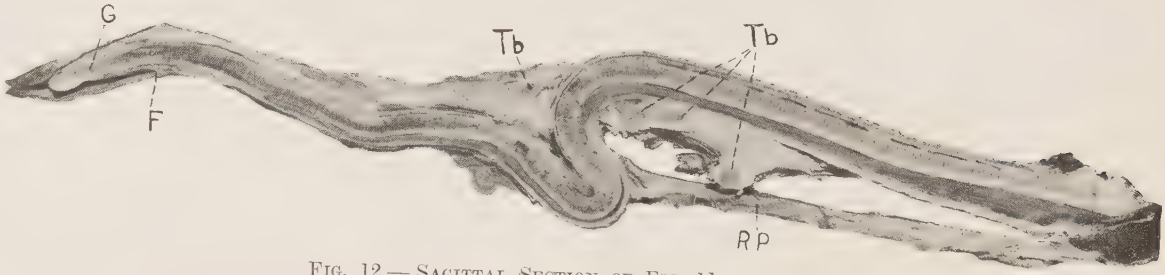

FIG. 12.— SAGITTAL SECTION OF FIG. 11.

G, glans; F, fornix; RP, retractor penis muscle, attached to second curve of sigmoid flexure of penis; Tb, tubercular masses about S-curve; A, tubercular abscess.

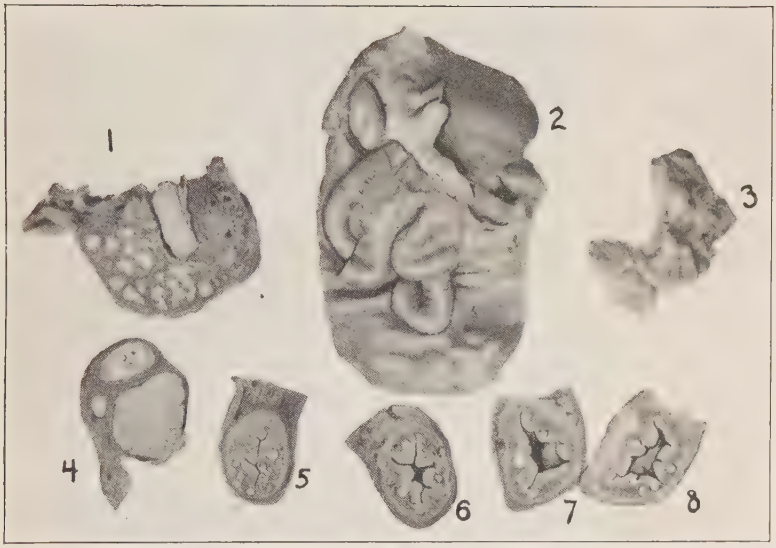

Fig. 13.— Tuberculosis of Ovary, Oviduct, and Uterine Walls.

1, section through oviduct, showing a mass of tubercles; 2, oviduct, enlarged and adherent, with adherent ovary shown at the upper left hand corner; 3, normal oviduct, for comparison; 4, ovary, showing above a tubercular mass, and below and to the right a corpus luteum; 5, 6, 7, 8, cross section of uterine horns, showing tubercles in submucosa.

FIG. 14.— SERIAL HORIZONTAL SECTIONS THROUGH OVARY AND OVIDUCT. SHOWING SEVERE NECROSIS AND ABSCESSATION OF OVARY AND OVIDUCT.

Ov′, section through greatly enlarged, abscessed ovarian end of oviduct; O, ovary; A, parovarian adipose tissue.

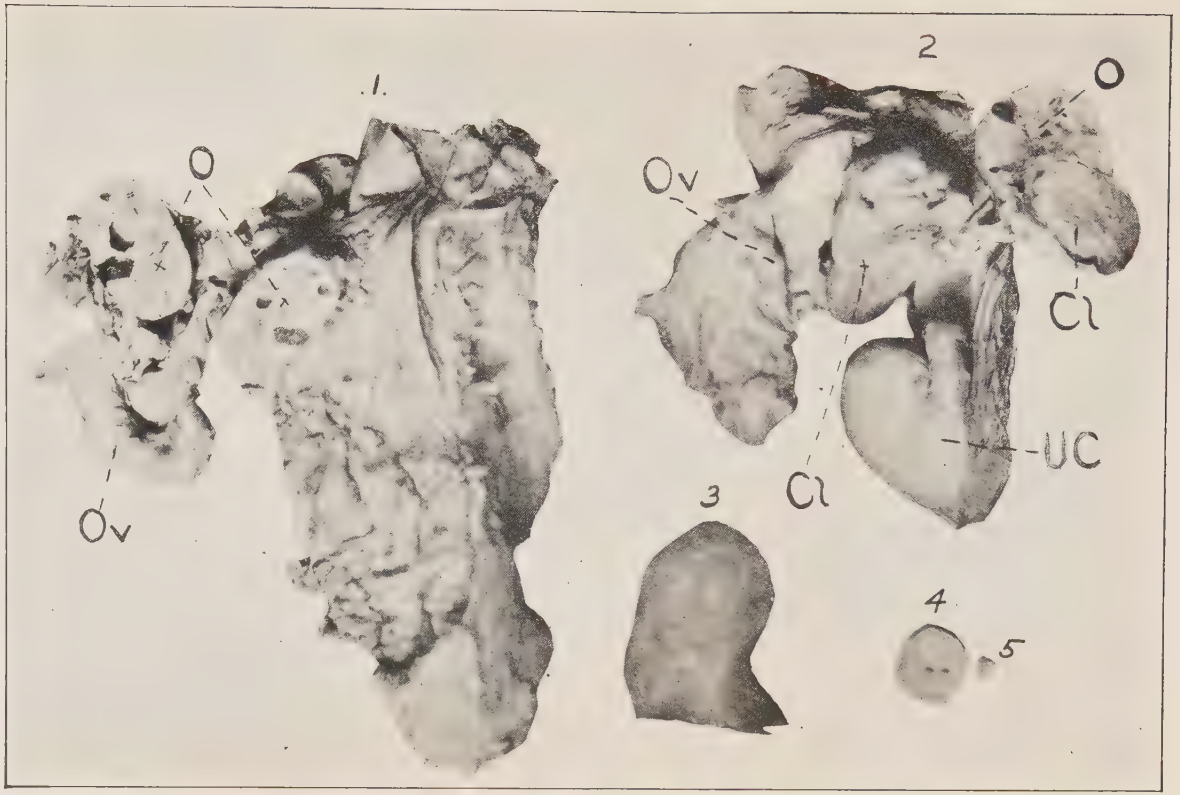

FIG. 15.— TUBERCULOSIS OF OVIDUCTS, BROAD LIGAMENTS, AND UTERINE CORNU.

O, ovary; Ov, oviduct; Cl, corpus luteum; UC, uterine cornu; 1, ovary (divided), oviduct and broad ligament; 2, ditto and uterine horn; 3, cross section of horn, showing faintly numerous tubercles in submucosa; 4, tubercular, and 5, normal oviduct, in cross section.

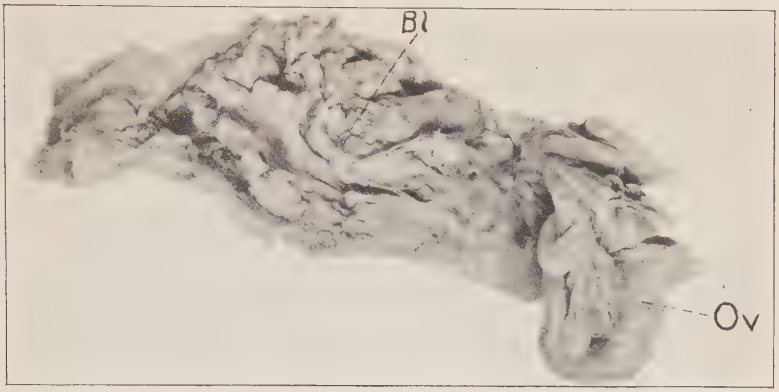

Fig. 16.— Tuberculosis of Broad Ligament.
Bl, broad ligament; Ov, oviduct.

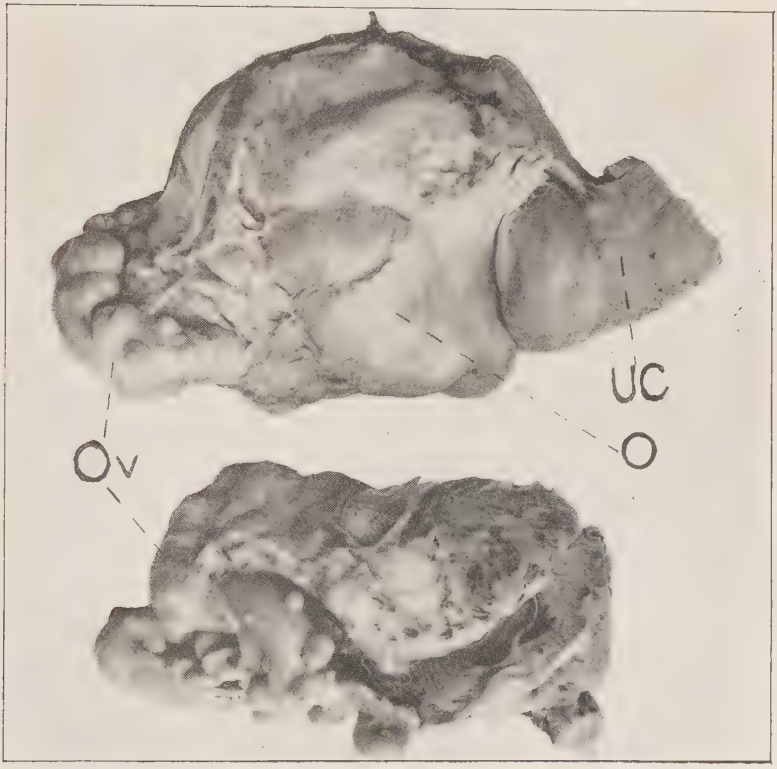

Fig. 17.— Tuberculosis of Ovary and Oviduct, the Oviducts Being Very Nodular and Enlarged, the Ovary Adherent in the Pavilion and Broad Ligament.

Ov, oviduct; O, ovary; UC, uterine cornu.

FIG. 18.— DORSAL SURFACE OF TUBERCULOUS OVIDUCTS, UTERUS, AND VAGINA, SHOWING EXTENSIVE PELVIC ADHESIONS AND ADHESIONS OF OVARIES IN PAVILION OF OVIDUCT AND BROAD LIGAMENT.

O, ovary; UC, uterine cornu; Ov, oviduct; U, uterus; V, vagina.

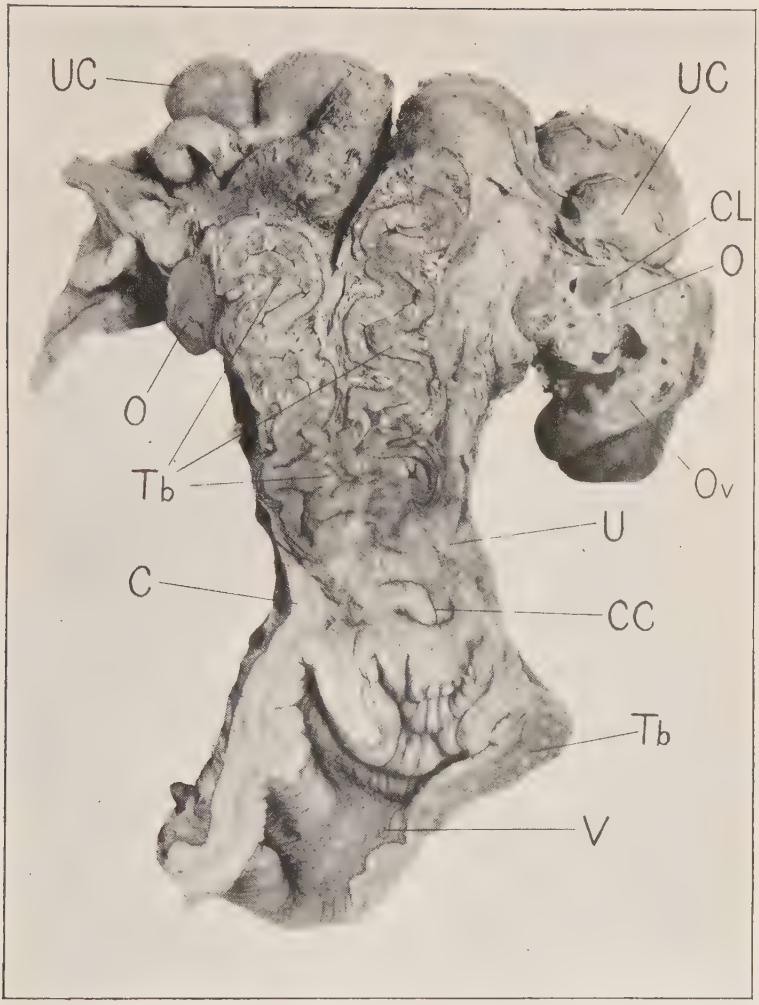

Fig. 19.— Horizontal Section of Fig. 18, Showing Tubercles, Tb, in Uterine and Vaginal Walls. The Right Oviduct, Ov, is Greatly Enlarged, Especially at its Ovarian End, Crossed by the Line From O.

CL, corpus luteum in right ovary, O; C, cervix; CC, cervical canal. Other lettering same as in Fig. 18.

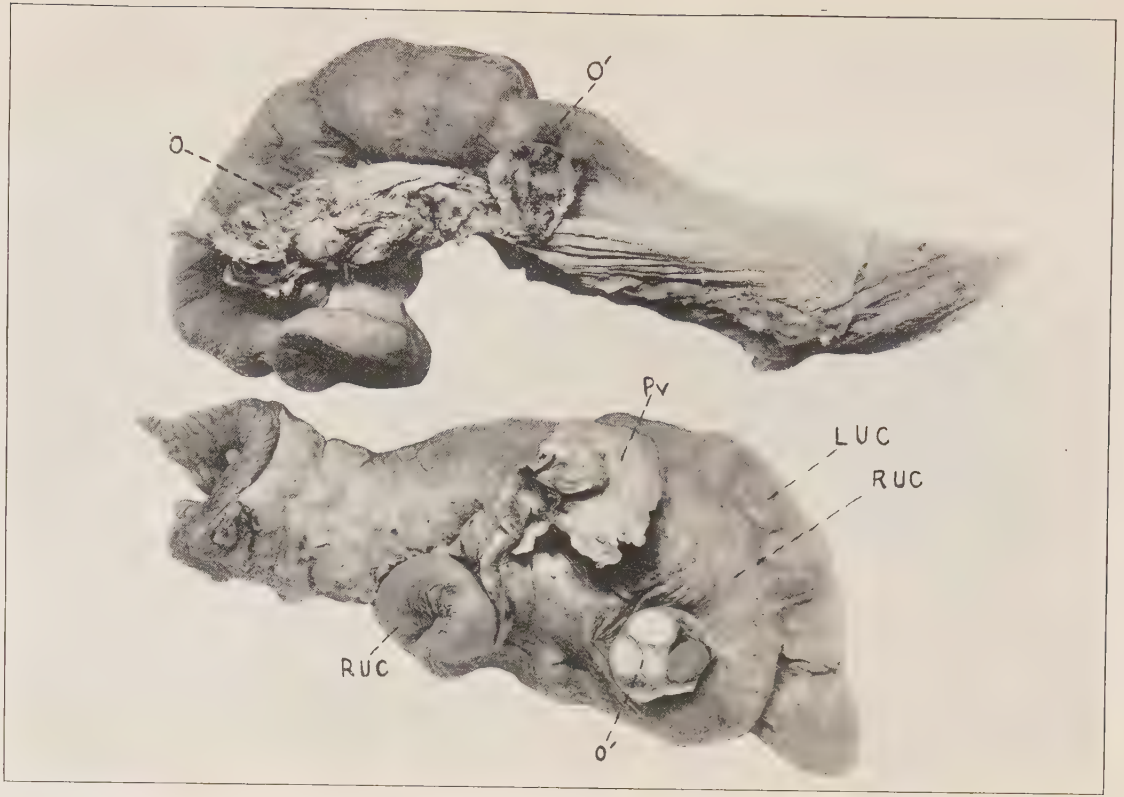

FIG. 20.— EXTERIOR OF SEVERELY TUBERCULAR UTERUS WITHOUT PELVIC ADHESIONS.

O, divided right ovary in situ; O', O', excised portions of ovaries lying in uterine cornu; Pv, pavilion of right oviduct spread out and covering remnant of right ovary; RUC, RUC, right horn; LUC, left horn.

FIG. 21.— SAGITTAL SECTION THROUGH FIG. 20.

1. tubercular pyometra, showing pus masses in cornual cavity; 2, pus masses in apex of cornu; CC, cervical canal. The corrugated uterine walls are everywhere thickly set with tubercles.

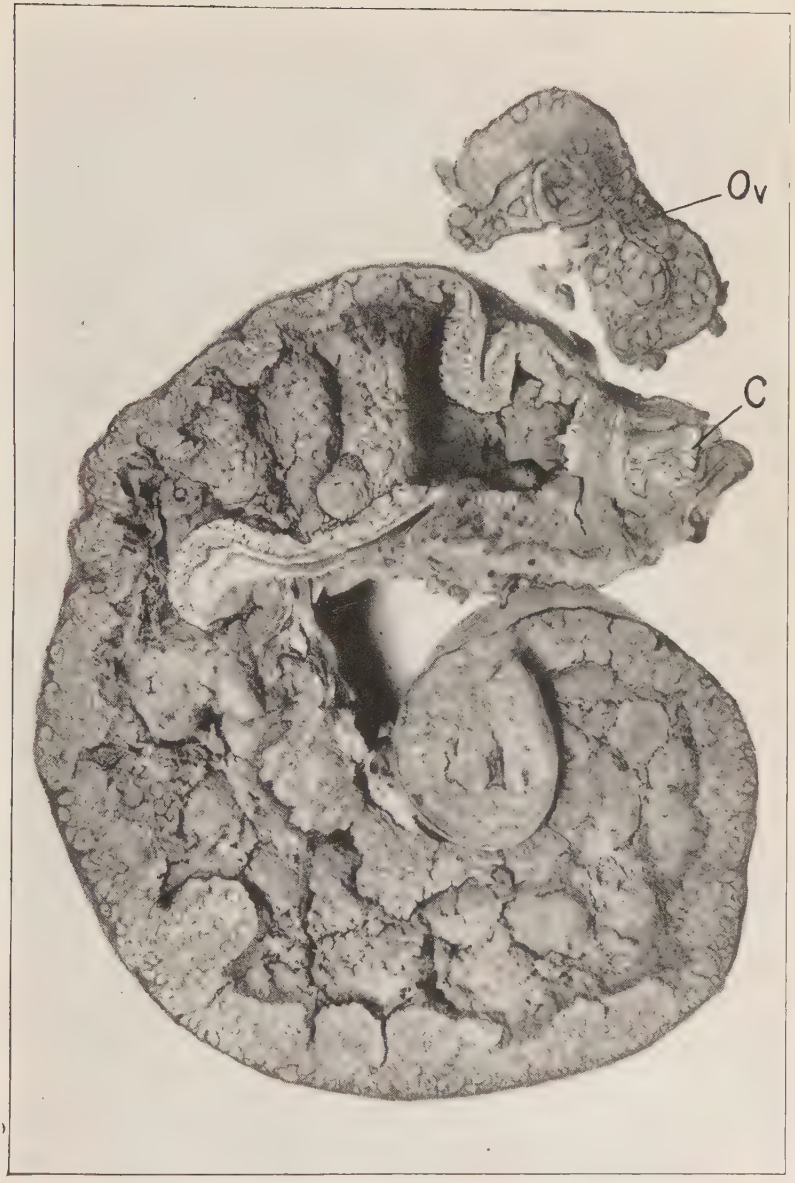

FIG. 22.— ADVANCED TUBO-UTERINE TUBERCULOSIS, WITH NECROSIS OF MUCOSA AND PYOMETRA.

Ov, Oviduct, greatly enlarged and consisting of a mass of turbercles; C, cervical canal.

FIG. 23.— MILIARY TUBERCULOSIS OF THE SUPERFICIAL UTERINE MUCOSA OF A VIRGIN HEIFER.

V, vagina; C, cervix; UC, uterine cornu; Ov, oviduct. The uterine horns are laid open to show the numerous small tubercles upon the mucous surface.

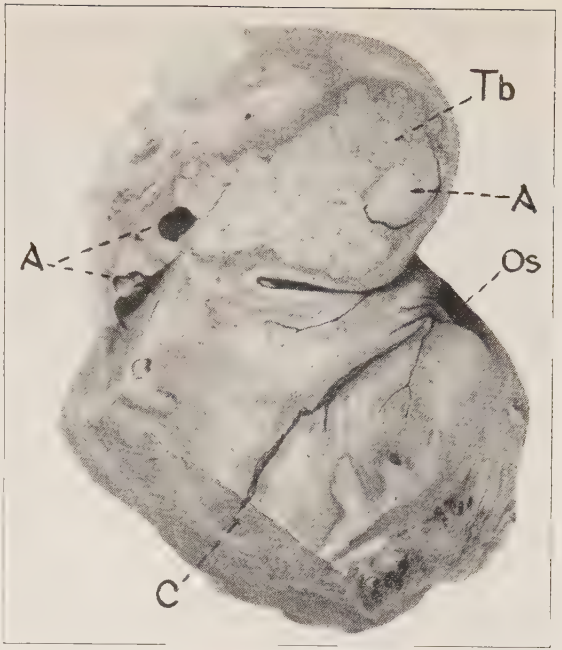

Fig. 25.— Sagittal Section of Fig. 24.

Os, os uteri externum; C, cervical canal; Tb, tubercles in lip of os uteri; A.A, tubercular abscesses.

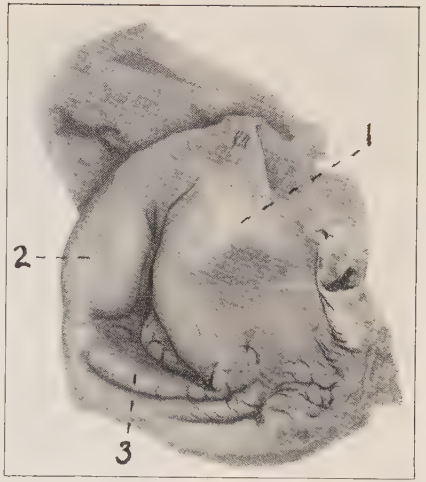

Fig. 24.- Tuberculosis of the Vaginal Portion of the Cervix.

1, greatly enlarged tubercular lip of os uteri externum; 2, a second, lesser tubercular tumefaction of lip; 3, mucous folds of the first annular cervical fold. Between it and 1 is the os uteri externum.

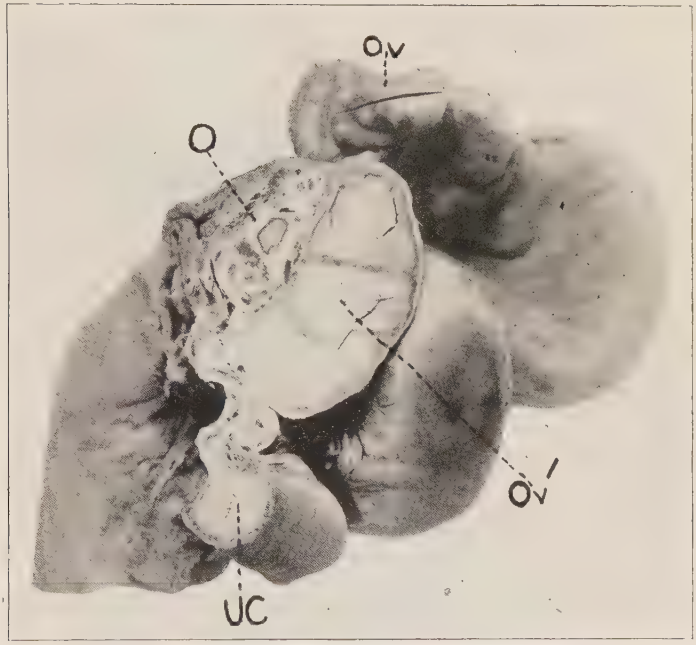

Fig. 26.— Uterus of Figs. 24 and 25.

O, bisected ovary; Ov, greatly enlarged and very nodular oviduct; Ov′, cystic ovarian end of oviduct; UC, cross section of horn.

become reflected upon the body of the penis and the continuity of the outer with the inner layer at the base of the glans serves as an unyielding ligament, holding the penis firmly in its retracted position.

Clinically, the presence of the lesion is probably not observed until well established. That is, the infection probably occurs without attracting notice, and, if opportunity is granted, probably several copulations are made during the few succeeding days, without attracting attention. When the lesion has reached a certain development, a given copulation, especially with a heifer, irritates the lesion severely and sets aflame the established infection. Then the lesion is observed, and is naturally attributed to mechanical injury in the last copulation, which is only partly true. Then swelling in the preputial region becomes evident, incarceration of the penis follows quickly, and copulation cannot occur.

The diagnosis is fundamentally dependent upon symptoms essentially identical with those seen in sheath tuberculosis, except that the lesion is located further backward, just anterior to the scrotum. It cannot be differentiated from other infections by an ordinary physical examination. Before an accurate diagnosis can be made, it is essential to include the tuberculin test and bacterial search. In a breeding sense, the lesion is not subject to cure.

(4) *Tuberculosis of the penial glands* is by far the the most commonly observed type of genital tuberculosis in bulls and sends more valuable breeding sires to the shambles than any other genital lesion, perhaps more than all other genital lesions combined. Apparently entering through a lesion, either recognizable or unrecognizable, of the glans, sheath, or prepuce, it may involve the lymph glands at any point from the margin of the sheath, along the prepuce, and up to and above the sigmoid flexure. Clinically, in opening the small peri-penial abscesses, I find them containing thick pus, the abscess walls dark, angry red, *without calcification.* The lymph glands lie chiefly along the sides of the penis. When involving the small glands outside the mucous membranes of the sheath and prepuce, the inflammatory adhesions soon prevent the reflection of these membranes upon the exterior

of the penis, make its protrusion impossible, and exclude copulation. Swellings in the region, usually well defined and painless, are evident. Abscessation gradually develops, but the abscesses are small, their walls sclerotic, non-fluctuant, and without a tendency to "point" or break. The lesions are well illustrated in Figures 5, 9, 11 and 12.

Sometimes the sigmoid lymph glands are involved, while those of the sheath and prepuce are slightly or not at all affected. In such cases, as shown in Figures 11 and 12, the chief clinical phenomenon is inability to protrude the penis and copulate. The inflamed glands induce adhesions which inhibit the elimination of the sigmoid flexure. Palpation of the region enables the veterinarian readily to detect the enlarged glands.

The enlargement of the lymph glands of the sheath, prepuce, or sigmoid flexure constitutes strong evidence of primary venereal tuberculosis. Other infections may induce similar adenitis, but commonly the veterinarian is justified in provisionally diagnosing such lesions as tuberculosis. He may, should circumstances demand, search for bacterial or other evidence. On the whole, any chronic adenitis of these glands ruins the breeding value of the bull and dictates slaughter. A positive diagnosis is highly important, even when made post mortem. If it is tuberculosis — and that is the rule — there must be highly dangerous cows in the herd, the genitalia of which should be subjected to very rigid search for tuberculosis. While a bull may become infected in copulating with a cow when the exterior of the vulva is contaminated with tubercle-bearing fecal excreta, this is improbable. The assumption should generally be that the infection has arisen from copulating with a female having tuberculous genital catarrh. Such an offender should be detected and eliminated. She is almost always incurably sterile. She constitutes a great peril to any breeding sire, and is one of the worst of "spreaders" of tuberculous infection.

TUBERCULOSIS OF THE FEMALE GENITALIA

Every part of the genital organs of the cow is subject to invasion. My collection contains examples of ovarian, tubal, uterine, cervical, and vaginal tuberculosis, and I have observed clinically vulvar tuberculosis.

(1) *Ovarian tuberculosis* is very rare, but is far more common than orchitic tuberculosis. The ovary is more exposed to the infection than the testicle, especially in the presence of extensive peritoneal lesions. I have obtained but two good examples. One of these, Fig. 13, is highly interesting, because casual examination suggests that the avenue of invasion of the ovary was through the physiologic lesion, the crater of a ruptured ovisac. The dense investing tunic of both the testis and the ovary appears to offer a highly effective barrier against tuberculous invasion, with the important difference that in the ovary there occur intervals (ovulation) when the tunic is ruptured and its continuity temporarily in abeyance. Viewed in this light, more ovarian than orchitic tuberculosis might be expected. Apparently this is true. Nevertheless, the ovary is highly resistant to the infection, as compared with other portions of the female genitalia. Frequently the uterus and oviducts are highly tuberculous, while the ovary remains normal. Generally, when the oviducts are involved, the pavilion is adherent to the ovary, and the peritoneal side of the pavilion is studded over with tubercles, as shown in Figures 15, 16, and 17, but the adherent, encapsuled gland resists invasion.

Ovarian tuberculosis cannot, in my experience, be directly and positively diagnosed clinically. Clinical diagnosis is not highly important, since ovarian invasion rarely, if ever, occurs without tubal and uterine tuberculosis, each of which is open to reasonably safe clinical diagnosis. So far as I am aware, ovarian tuberculosis induces no clinical symptoms. There is a definite impression given in veterinary literature that ovarian tuberculosis causes sterility at times, but there is no evidence that it plays an essential role. So far as I have seen, it is the coëxisting tubal and uterine tuberculosis which causes the sterility. Statements occur also (Hutyra and Marek, Law) that genital tuberculosis, either through the invasion of the ovaries or otherwise, induces nymphomania. No evidence is submitted upon the point. The power of tuberculosis of any portion of the genital tract to cause nymphomania is probably pure legend. A careful study of nymphomania shows it to be due to a definite type of cystic degeneration of the ovary, wholly devoid of any trace of relation to tuberculosis. Genital tuberculosis and the nymphomaniacal type of ovarian

cyst may coëxist, but that is not evidence of either identity or relationship.

(2) *Tubal tuberculosis* is, next to uterine tuberculosis, the commonest type observed in the genitalia of cattle. In many specimens, the appearances suggest that the infection has invaded the oviducts centrifugally from the peritoneal cavity through the pavilion. In other instances of even severe uterine tuberculosis, the oviducts are free. Tuberculous oviducts are usually recognizable by rectal palpation. They become enlarged and very hard. Generally they are adherent and studded over with large tubercles. The tuberculous tubes vary in transverse diameter up to 0.5 inches or over. When much enlarged, they become elongated and thrown into folds lying in front of and lateral to the ovary, as shown in Figures 18 and 19. The disease may be confused with ordinary pyosalpinx or hydrosalpinx. In ordinary pyosalpinx, there are two types. In the first and commonest, there is not much pus and the oviducts are not greatly enlarged, but are very firm and of even contour. In the second type of pyosalpinx, the pus is voluminous and the oviduct walls attenuated, giving a soft fluctuating tube, with thin, atonic walls. In hydrosalpinx, the oviduct is distended with lymph, firm and fluctuant. Tuberculous salpingitis is, in my experience, always nodular, the tubes very hard, adherent, and quite large.

Tuberculosis of the oviduct is almost, if not always bilateral, rendering the animal incurably sterile as well as a peril in a breeding or milking herd. The pus from tuberculous pyosalpinx escapes through the uterus and cervix to the vagina, to constitute a very serious menace to the bull.

(3) *Uterine tuberculosis* is the commonest type of genital tuberculosis in cattle. It is the most dangerous type of genital tuberculosis. The peril of uterine tuberculosis to other cattle is double. It is supremely dangerous to the bull when copulating with the affected animal. The discharges resulting from the tuberculous uterine catarrh soil the vulva, tail, thighs, and ultimately the udder, and contaminate the milk. Thus the milk becomes intensely dangerous for calves, for other animals, and for man. In its peril to animal and human health, uterine tuberculosis vies

with tuberculosis of any other organ. Uterine tuberculosis is quite variable, showing three somewhat separable types:

(a) Peritoneal uterine tuberculosis with extensive pelvic adhesions, as shown in Fig. 18.

(b) Tuberculosis of the glandular structure of the uterine mucosa, as shown in Figures 21 and 22.

(c) Tuberculosis of the epithelial layer of the mucous membrane, as shown in Fig. 23.

No clear line of demarcation can be drawn between the three types, but in most cases one of the three groups of lesions predominates.

In uterine tuberculosis there is generally a persistent, obstinate uterine catarrh. In some cases the catarrhal discharge is inconspicuous. Douching the uterus may reveal very little pus. In some cases of tuberculous uterine catarrh, uterine douches affect the catarrh favorably for a time, but permanent relief I have not observed. In cases where there have been apparently favorable results from douching, misleading the practitioner as to the nature of the malady, limited observation indicates that copulation sets the infection aflame. In other cases, such as Figures 21 and 22, the catarrh is profuse, but not generally fetid. Like tuberculosis of the oviducts, uterine tuberculosis is usually, if not always symmetrical, or bicornual. Generally the uterus contracts extensive pelvic adhesions. In many cases the peritoneum is so involved that ovaries, oviducts, uterus, and broad ligaments constitute a complex adherent mass, as in Fig. 18, where it is difficult, though generally possible, to identify the various parts by rectal palpation. Pelvic adhesions due to genital tuberculosis resemble strongly pelvic adhesions due to other infections. The adhesions due to tuberculosis are often accompanied by palpable tubercles. Their recognition aids materially in diagnosis. Non-tuberculous pelvic adhesions are frequently accompanied by sclerotic abscesses of an ovary, oviduct, uterine horn, or the three areas combined. In my collection, no such condition exists in any case of tubo-uterine tuberculosis, not even abscessation of the lymph glands in the broad ligaments of the uterus. Tuberculous pyosalpinx and pyometra, with necrosis of the uterine mucosa, as indicated in

Figures 21 and 22, is common enough, but I have not observed extensive tuberculous abscesses involving the ovaries, oviducts, uterus, or broad ligaments. There is one important difference in the clinical history of tuberculous and non-tuberculous pelvic adhesions. The non-tuberculous adhesions are commonly preceded by acute metritis, largely with placentitis and retained fetal membranes. Tuberculous pelvic adhesions ordinarily arise independently of acute metritis.

The cow from which Figures 18 and 19 were taken was entered in our clinic for sterility. There was slight uterine catarrh (tuberculosis of the uterine mucosa) which abated under antiseptic douches. The ovaries, oviducts, and uterus were normal by rectal palpation. She appeared so well that she was mated with a valuable herd sire. Soon afterward, she broke down, and upon re-examination genital tuberculosis was evident. When destroyed, extensive generalized tuberculosis was revealed. As she entered our clinic without a history, but upon the assumption of freedom from tuberculosis, no careful search was made in that direction. Apparently the disease reached its crisis at about the time of copulation, the patient gave way to the infection, and a diagnosis of genital tuberculosis became practicable. Fortunately the sire escaped, largely perhaps because the cow was douched just prior to copulation. The case illustrates vividly the insidious and subtle manner in which genital tuberculosis in the cow may develop and how the veterinarian needs be on his guard.

In some cases the tuberculous uterus remains to the end free from adhesions, as in Figures 20, 21, and 22. It is a significant fact that in my collection those uteri in which the mucosa is most extensively involved and which have undergone the maximum increase in size have suffered least from pelvic adhesions. Is this because in such cases as Figures 20 and 22 the infection of the uterine cavity has been primary (venereal) and in those cases with extensive adhesions, as in Figure 18, the infection has invaded the tract by continuity from the general peritoneum? The freedom of the oviducts in Figure 20 appears to emphasize this suggestion.

When the uterus is enlarged and sclerotic and comparatively free from adhesions, the tuberculous character is difficult of clinical

recognition. One may meet with similar sclerotic hypertrophy in actinomycosis (primary genital) and in chronic purulent sclerotic metritis with destruction of the uterine mucosa. In such cases however, the outlook for the breeding life of the patient is hopeless and an accurate clinical diagnosis is not imperative. In some cases of uterine tuberculosis, the masses of tubercles are so voluminous that they throw the uterine walls into great transverse folds which may be palpated per rectum. When such folds are present, they have a distinct diagnostic value. Usually in chronic abscessation of the uterus due to ordinary pyogenic bacteria or to actinomycosis, while the uterus is enlarged and hard, it is very irregular in outline. At one point there may be little or no enlargement. In actinomycosis and in pyogenic abscessation of the genitalia, some one part usually forms a great abscess or abscesses quite overshadowing other lesions, but in tuberculosis there is a strong tendency to symmetrical enlargement. The two horns are approximately alike in volume, form, and consistence. This is important to remember in making a diagnosis. In one cow of great breeding value, a reactor to tuberculin, which had not calved for two years and had long suffered from an abundant, fetid, highly repulsive genital discharge, I found one uterine horn much enlarged, three to four inches in its transverse diameter, and irregular in its contour. The other horn was approximately normal. The findings by palpation, in conjunction with her reaction to tuberculin, led me to diagnose uterine tuberculosis. The autopsy revealed, instead, a macerating fetus, the bones of which caused the irregular bulging of the cornu. Had I recognized the fact that in uterine tuberculosis the enlargement of the horns is usually symmetrical and that the pus from the tuberculous uterus is not usually fetid, the error would probably have been avoided. In those cases of uterine tuberculosis where only the superficial mucous layer is involved, as in Fig. 23, the diagnosis by ordinary clinical examination fails. The ovaries, oviducts, and uterus are normal to palpation, the uterine catarrh is insignificant, and no outstanding clinical evidences of genital tuberculosis are present.

The outstanding elements in the differentiation of utero-tubal tuberculosis are the pelvic adhesions, the tubercles in the genital peritoneum, the symmetrical enlargement of the uterus, and the

obstinate uterine catarrh. No one of them warrants a positive diagnosis. If the patient reacts to tuberculin, the probability of genital tuberculosis is increased. No data are at hand regarding bacterial search of the genital discharges. Such search would probably aid materially in diagnosis. A negative tuberculin test is not conclusive evidence that genital tuberculosis is not present. The very valuable herd bull from which Figure 5 was made quite certainly contracted the infection from a cow which had successfully passed several tuberculin tests, in spite of severe utero-tubal and general tuberculosis, with tuberculous uterine catarrh. While in many cases, such as delineated in Figure 18, quite an accurate physical diagnosis of genital tuberculosis may be made, not all cases may be detected. The important point is that utero-tubal tuberculosis may almost always be tentatively diagnosed by rectal palpation and the veterinarian and owner placed on guard. So long as the symptoms named are present, the tentative diagnosis of utero-tubal tuberculosis is not only justified, but obligatory, from the sanitary standpoint. When uterine catarrh is present, copulation is contra-indicated by every consideration of hygiene. Fertilization can not occur; coitus aggravates the catarrh and imperils the bull. The retention in a dairy of a cow with obstinate uterine catarrh is unjustified by every consideration of health and decency. The afflicted animal is unfit to associate with others in such a manner that their food may become contaminated with the genital discharges.

A tentative diagnosis of utero-tubal tuberculosis works no serious injury. If the cow recovers, the recovery automatically corrects the diagnosis; if she fails to recover, she is worthless for breeding or dairying, and fit only for slaughter, whether the infection be tuberculous or otherwise.

(4) *Tuberculosis of the cervix* is presumably rare. I have but one specimen (Figures 24 and 25) derived from the abattoir, and hence without a clinical history. In this case, the uterus and oviducts are also involved. The illustrations suggest the probable clinical features. The greatly swollen, hard, smooth lips of the cervix are apparent, and could not well be missed by an expert examining the genitalia clinically. While uterine catarrh was evidently present in this animal, it presumably had nothing to do with the cervical lesion. The lesion was apparently closed and caused

no discharge. The surgeon, meeting with such condition, would logically incise the tumor, laying bare the nature of the lesion. I have repeatedly opened cysts in these parts. In so doing, I always draw the cervix back into the vulva before operating, so that the field is freely open to view and the contents which may be released are readily seen. If purulent, they at once raise the question of tuberculosis, which, once raised, should not be dismissed until the diagnosis has been made perfectly clear. On no account should a cow be permitted to copulate when a cervical lesion exists, unless it is clearly shown to be non-tuberculous. Even harmless-appearing cysts should not be passed over carelessly. Like vulvar cysts, discussed below, they may be tuberculous. The cervical lesion, when open, is evidently extremely dangerous.

(5) *Vaginal tuberculosis* is apparently very rare. Figure 19 illustrates the only specimen in my collection. It was not recognized clinically, although I examined the patient several times. However I did not search for vaginal tuberculosis. Possibly it was of recent origin.

It is important for the veterinarian, in dealing instrumentally with diseases of the genitalia, to bear in mind that he may, by the use of the uterine forceps, implant the infection in the cervix, or in the vagina at its point of continuity with the cervix. He may re-infect these parts, through instrumental lesion, with the discharges from the patient's uterus. If careless in his methods, he may carry the infection from a tuberculous cow to a healthy one. Under reasonable precautions, the danger is quite negligible. Figure 19 shows that in this case the tuberculous lesions are within the vaginal wall and closed. If open, the lesion would have the same danger as that of the cervix.

(6) *Vulvar tuberculosis* has occurred twice in my practice, each time readily diagnosed, though in one case the nature of the lesion was at first misleading. In this case, at the first examination, a cyst one inch in diameter lay in the vulvar lip and was incised just at the labial margin, and a small amount of seropurulent content escaped. Prior to my next examination, the patient had reacted to the tuberculin test, and when I next examined her, the lesion presented the typical characters of a

tuberculous ulcer. My second case was more readily diagnosed. The vulvar lip, which was swollen and sclerotic, presented an old fistulous opening, from which small volumes of thick pus could be pressed out. Centripetally, a tuberculous gland, two to three inches in diameter, lay alongside the vagina. In addition, both supramammary glands were greatly enlarged, and at each internal inguinal ring glands three inches in diameter were palpable. Autopsy showed severe generalized tuberculosis.

Vulvar tuberculosis is evidently a *very* dangerous type for the bull. In my second case, the cow had been bred to the herd bull, valued at more than ten thousand dollars, but a few days before my examination. Fortunately he escaped infection.

Having regard for the facts set forth, it seems remarkable that veterinary writers have given so little attention to the subject of genital tuberculosis in cattle. The failure to give the subject due consideration is apparently due to two circumstances:

(1) The average veterinary practitioner fails to diagnose the disease in either sex.

(2) In the abattoir, the genitalia are not ordinarily inspected.

In the bull, the preponderant types disable the patient for coition and he must be sent to the shambles, regardless of the nature of the disease. He is impotent, and ceases to be of value as a sire. The fact that a highly dangerous cow lurks somewhere in the herd is not recognized. The cow with utero-tubal tuberculosis is sterile, and beyond that fact the practitioner fails to go.

Careful study must make it apparent that genital tuberculosis is not rare and that it offers one of the most, if not the most dangerous train of lesions observed in any form of tuberculosis. Most types of genital tuberculosis in the bull are highly dangerous to cows with which he may copulate, so long as he is capable of copulation, but the bull often becomes quickly incapacitated sexually and the special venereal dangers are placed in abeyance.

The cow with genital tuberculosis, be it tubal, uterine, cervical, vaginal, or vulvar, is intensely dangerous. I have seen more valuable breeding bulls sent to the shambles from serving such cows than for any other disease, or for all other diseases combined. The genital discharges from open lesions find their way inevitably along the tail, thighs, and escutcheon over the udder to the teats. There it is taken with the milk when a calf sucks, or the milk is

contaminated as it is drawn, and finally consumed by calves or people.

The control of these dangers evidently depends chiefly upon the more general and accurate diagnosis of genital lesions in both sexes and the more general adoption of efficient sex hygiene in breeding cattle. The lesions are to be detected through two chief agencies — dairy inspection and inspection of the genitalia in connection with sterility. At present, dairy inspection fails utterly in most cases in relation to genital disorders. Repeatedly I find, in regularly inspected herds, and even in certified dairies, cows with quarts or gallons of fetid pus in the uterine cavity, large volumes of which are pressed out from time to time while the cows are lying down. Advancement in this field is one of the most urgently needed reforms in dairy inspection. The greatest weapon for the control of the danger lies in the more general inspection of the genitalia of cattle, as a safeguard against sterility and abortion. It is becoming more and more apparent that a careful physical examination of the genitalia of cows, prior to breeding, is a wise precaution of distinct scientific and economic importance. It is beginning to dawn upon some progressive breeders of pedigreed cattle that, before permitting the herd sire to serve a cow, her genitalia should be subjected to a careful inspection and that she should not be bred until it is determined, so far as possible, that she is sound. A study of the problem indicates further that the breeder should take far greater care in the sex hygiene of the bull. If penial injuries and infections are to be averted, having first ascertained that the genital organs of the cow are healthy, the external genitalia of both cow and bull should be carefully washed before permitting copulation. It is highly important, in the prevention of penial lesions, which may serve as infection avenues, that the crusts of pus in the vulvar tuft of hairs of the cow be removed by washing. Otherwise, these crusts of pus may be caught and forced into the vulvar opening, to abrade the penial mucosa. After copulation, the penis, prepuce, and sheath of the bull should again be carefully and efficiently douched. These precautions go hand in hand with those designed to control sterility and abortion.

THE ETIOLOGY AND MODE OF INFECTION IN WHITE SCOURS OF CALVES

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The disease of young calves known commonly as white scours, or calf scours, is one from which probably few localities of the United States are entirely free. In some, especially those districts given over to dairying, it occasions great losses. In these districts, it is readily recognized by the farmers, and takes its place, not far behind contagious abortion and tuberculosis, among the demoralizing diseases of the dairy industry. In some places the disease is of the acute type and the calves die within a day or a few days after birth with symptoms of intoxication and profuse diarrhea. Sometimes the intoxication is so great, and so rapid, that apparently the diarrhea does not have time to develop and the calf dies without showing diarrhea. Sometimes the disease is less acute and the scouring may be negligible, while joint lesions and the peculiar type of pneumonia which accompanies the disease may predominate. In these cases the calves frequently die after a prolonged illness, and if recoveries do occur the animals are usually stunted and unthrifty.

In view of the importance of the disease, it is quite surprising, when looking over the literature, to find how little has been recorded in the English language, especially in this country. Most of the accounts of the malady which have appeared here seem to have been drawn largely from the German or the Danish or from the work of Nocard (1) in Ireland. The references to the work of the latter author we believe to be unfortunate, since a careful study of the disease, as he reports it, leads us to the conclusion that it was materially different, with a different etiological agent and mode of infection from the disease which we have in this country. In some localities Nocard's disease may exist, but it is not the one commonly found. The "Kalberruhr" of continental European investigators appears to be the same as our disease.

Historical

White scours in calves has been known for more than a century. The first good description was that given by Tolnay in 1799. He described the condition as due to the coagulation of milk in the fourth stomach (a purely physiological condition) together with an increased acidity of the intestinal contents. From this time until the work of Obich was published in 1865, the disease was always supposed to be due to dietetic errors. Obich brought forth the infection theory and stated that the infectious material existed in the diarrheic feces. Franck (1876) held the view that the cause of the disease was a miasma which lived and developed in the floors of the stalls.

Dieckerhoff held that the infection occurred orally in the taking of nourishment soon after birth, and agreed with Franck that the causative agent lived in the floors and could maintain itself there all year long.

Friedberger and Fröhner thought it probable that the infection really occurred during birth from an infectious vaginal catarrh. According to this view, the infection could enter either through the mouth or through the wound produced by the rupturing of the umbilical cord.

Nocard (5) (1866) went still further and expressed his belief that the infection occurred within the uterus. The main reason for this belief was the exceedingly brief interval which existed frequently between birth and the appearance of the first symptoms. We have found no experimental data bearing upon the subject.

The first bacteriological study of the disease was made by Jensen (6) of Copenhagen in 1891. He found in the blood and internal organs of calves dead from the disease pure cultures of the colon bacillus — an organism, or group of organisms, less understood then than now. Feeding this organism to new-born calves in their milk, he produced the disease in its typical form. Subcutaneous inoculations resulted either in a localized abscess or a rapidly fatal septicaemia without evidences of scouring. Feeding experiments with cultures derived from the feces of normal calves resulted negatively, showing that the type which was found in the scours cases was either a pathogenic strain or an ordinary strain, which for some reason had acquired patho-

genicity. From these studies, Jensen concluded that calf scours was caused by members of the colon group, which gained entrance to the calf through infected food, straw, or surroundings which the animal might lick.

Piana (1895), like Jensen, succeeded in finding the colon bacillus in these cases, but was unable to produce the disease with the cultures obtained.

During the same year, Monti and Veratti (7) published the results of some work upon the disease, in which they considered B. coli the etiological factor.

Almost simultaneously with the preceding works, there appeared the results of the researches of Mazzanti and Vigezzi (8) in Italy. These workers considered an oval diplococcous, which they isolated from the liver, intestine, brain, and vessels of the umbilical cord, as the cause of the malady. They declared the disease due to an umbilical infection.

In 1899 there appeared the work of J. Poels (9) of Rotterdam, on diseases of sucking calves. This dealt largely with calf scours and gave the data collected on a large number of cases. Unlike former investigators, Poels failed to find a constant organism, and accordingly grouped the cases coming under his notice according to their etiological agents. He considered the disease as a septicaemia, and thought the final result the same, whether the infection occurred through the navel or through the digestive tract. His grouping of the diseases which he considered as coming under the general head of calf scours is as follows:

(1) General infection with the colon bacillus.
(2) General infection with streptococci.
(3) General infection with colon and streptococci.
(4) General infection with pseudo-colon organisms.
(5) General infection with pseudo-colon and streptococci.
(6) General infection with *Ps. pyocyaneus.*
(7) Intoxication from local infection with *B. proteus.*
(8) Septicaemia haemorrhagica.
(9) Polyarthritis specifica.

Of all these forms, Poels considered the first, fourth, and sixth true intestinal infections, and all the others navel infections or complications of intestinal and navel infections.

In the results of their work, published in 1901, Lesage and Delmer (10) of the Pasteur institute incriminated a coccobacillus of the Pasteurella type which they were able to isolate by the inoculation of rabbits with blood and also with feces. They failed to produce the disease with this organism. The colon bacilli which they found on cultures made post mortem they attributed to agonal invasion from the intestinal tract.

The work on this disease which has attracted the most attention among English-speaking people is that of Nocard (1, 2, 3) in 1901 and 1902. In the spring of 1901, at the request of the Irish Board of Agriculture, he went into the western part of Ireland to conduct an investigation upon the destructive calf disease which existed there. After a very short time in the district, and the study of a very small number of cases, he made a report which was quite generally accepted by English and American veterinarians. He declared the disease due to an organism of the hemorrhagic septicaemia type which gained access to the body of the calf through the unhealed navel stump, and outlined a method of prophylactic treatment which had as its basis the disinfection of the navel stump. It was Nocard who gave to the disease the name "white scours", and first called attention to the joint lesions and the "lung disease" which so frequently accompanied it.

The work done during the last fifteen years on the disease does not appear to have been very fruitful. Nothing essentially new has been learned. In recent years, references to the disease may be found occasionally in American literature, generally in connection with contagious abortion of cattle. To Williams (11) belongs the credit of first calling attention to the fact that the disease runs more or less parallel to contagious abortion, and evidence is accumulating which tends to show that a close connection probably exists between the two diseases.

Etiology

Though much work has been done on the etiology of the disease by the various investigators mentioned, and a number of others, it is still in a very unsatisfactory state. At the present time, white scours is generally ascribed to the activities of the colon

bacillus or members of the colon-typhoid intermediate group. While these organisms have been found constantly in these studies in New York and the feeling is strong that they are responsible for much of the actual damage accomplished, not only to the calf, but also to the dam, still there are points about the hypothesis which throw a shadow of doubt upon these types as the primary invaders. Whether primary or secondary factors, the part they play in the disease is undoubtedly of great importance. One of the best proofs of this is the very beneficial therapeutic effects of white scours serum. This is a serum produced, as a rule, by the immunization of a horse or a cow to a number of strains of colon bacilli. In typical cases of the disease, in which navel complications have not occurred, and especially the acute cases in which profound organic lesions have not developed, this serum is of great value when given in doses large enough to combat the infection. Since this treatment is aimed at the invaders of the colon type only and so far as we know has no effect whatever upon any other agent, we must concede that the colon organisms are playing an important part in the disease.

In all the calves dying from white scours upon which an autopsy was held in this district, there has been no trouble in isolating B. coli from the blood and practically all organs, as well as from the contents of the digestive tract. In no case was a mixed culture obtained in these cases from the organs except when an umbilical infection existed simultaneously. Although at present there are not sufficient data to prove this point, it is thought that this may prove to be universally true. This would explain most of the groupings as given by Poels. One such case of umbilical complications with acute scours has come to my attention. In this case the calf was scouring profusely and was very dull. In addition, there was a painful, purulent arthritis in the right anterior fetlock joint, a suppurative navel, and purulent mastitis in one quarter of the udder. The earlier history of the calf was not known, but it is presumed that the udder was infected by some other calf sucking first the infected and discharging navel and then this teat, as young calves are apt to do, especially after feeding, if allowed to run together. Examination of the feces showed, as usual, almost a pure culture of B. coli

communior. Cultures from the navel and udder revealed M. pyogenes aureus and albus and Bact. pyogenes. Pus aspirated from the joint gave only Bact. pyogenes. Blood culture gave only M. albus.* Unfortunately, this calf died at a time when it was impossible to hold a post-mortem, but it is a certainty that, in this case, the colon organism would not have been recovered in a pure culture. It was simply a case of septicaemia of both navel and intestinal origin, and cultures from the blood, after death, would probably have yielded cocci, as well as colon bacilli.

In two cases of scours of the more chronic variety in the experimental herd, inflammation of the hock joints developed, as frequently occurs in the disease. The calves suddenly became lame and the joint capsules became distended. The joints were extremely hot and painful. Aspiration of the contents revealed a cloudy, limpid fluid. On cultures, both yielded B. coli communior in a pure state. One of these calves died the following day. Cultures made from the blood in the jugular vein, heart blood, lung, liver, spleen, and mesenteric lymph glands showed a pure culture of B. coli communior. Media inoculated from the umbilical vein remained sterile.

Hutyra and Marek state that B. coli communis is the variety most often found in cases of white scours. This has not been true in the cases studied in this vicinity.

Jensen claimed that he was able to produce the disease in a typical form by feeding to very young calves such drugs as creolin, pyoktanin, etc. He explained these results by saying that the resistance of the intestinal wall is reduced, allowing the ordinary colon types to migrate into the tissues, where they take on new virulence. After death, the tissues were found to be swarming with B. coli, which proved to be virulent to other calves. Feeding new-born calves with boiled milk instead of colostrum resulted similarly. Jensen used this experiment as an argument in favor of the hypothesis that the ordinary colon types took on virulence, instead of there being a distinct pathogenic strain, as argued by Poels.

In referring to Nocard's work, mention was made of the

* This was probably skin contamination. It requires extraordinary precautions, in disinfecting the skin of cattle, to prevent contamination of the culture with this micrococcus.

pneumonia which accompanied his white scours. According to his description of this condition, it was found on the same farm as scours, and practically all calves that recovered from the latter came down in six to ten weeks with this "lung disease". The following is Nocard's description of a case:

"Although lung disease is rare in April, we had the opportunity to examine one of the most characteristic cases in a six-weeks-old calf which a few days after birth had had a temporary diarrhea and which had shown no other symptoms except a lack in general condition, some cough, and labored breathing. The owner said he had lung disease. (In three years he had lost more than one hundred calves, and he knew by long and sad experience that the few subjects that seemed recovered from white scour die of lung disease when they are about two months old.) The animal was killed. He had an enormous pulmonary lesion; the posterior two-thirds of the left lung formed a compact mass, very heavy; on its surface, which was here and there bosselated and fluctuating, the pleura was thickened, covered with vegetations, and adherent by fibrous bands to the diaphragm and to the hypocardia. The tissue of the lung was the seat of an extensive sclerosis and hollowed with numerous cavities filled with thick grumous pus of a dirty white color, resembling mortar."

In these cavities of mortar-like pus, Nocard found a polymorphous organism which seemed to be identical with the organism of caseous lymphadenitis of sheep and ulcerative lymphangitis of horses — the bacillus of Preisz.

The form of pneumonia known as calf pneumonia, which accompanies scours in this country, is not at all like Nocard's type. The form that occurs here is not often extensive, but is confined usually to the lower part of one or both anterior lobes. It is a type of broncho-pneumonia, but rarely, if ever, does tissue destruction progress to the point of cavity formation. Sometimes the pneumonic foci extend and coalesce, forming larger areas which may form a lobar pneumonia (speaking in terms of the extent of the process) but as a rule comparatively small areas are involved. Cultures made from these lungs show colon bacilli in the great majority of cases. Bacterins made from these cases prove very efficacious in treating infected herds, especially when used in the early stages of the condition.

All of the facts given heretofore tend to strengthen the belief that white scours and pneumonia of calves are due to the invasion of the tissues with the colon bacillus. Several other facts tend greatly to weaken this conclusion.

A calf which is very ill with the acute type of calf scours shows all the symptoms of a profound septicaemia or toxaemia. The extremities are cold, the pulse and respiration greatly accelerated, the temperature commonly as high as 106 degrees F., there is a nasal discharge, and the animal is usually dull and listless, and so weak that it can not be made to stand. It seems in these cases that the colon organism, if it is responsible for the condition, should exist in the blood stream. Lesage and Delmer (10) state that if the animal is killed at this stage, before the death agony begins, the blood and organs show no colon bacilli. If the calf is allowed to die naturally, or is slaughtered as death approaches as the result of the disease, colon bacilli are found everywhere in large numbers. From these findings they concluded that Jensen, Poels, and others were dealing with a pure saprophyte which was released from the intestine when the vitality of the calf was sufficiently reduced just before death, and which had no significance in the disease. That the colon types may do this under certain conditions seems quite certain. If the colon bacillus is the actual primary invader in the disease, it is quite difficult to explain the cause of the toxic symptoms. B. coli does not produce any extra-cellular toxin, as has been repeatedly proven by various investigators by injecting filtrates of cultures into rabbits. This I tried myself by injecting a very young calf with a comparatively large quantity of a filtrate from a culture of B. coli derived from a white scours case. There was absolutely no reaction. The same culture, unfiltered, had been injected into another calf the previous day. A rapidly fatal septicaemia developed, and the colon organism was recovered from the blood and all organs after death.

Both septicaemia and toxaemia apparently having been eliminated, how may we account for the symptoms? This is a question which remains to be answered. That an ultra-microscopic virus might exist, Jensen suspected, but ruled it out by filtration tests. This work needs to be repeated, and will be the subject for further study.

The small amount of work which has been done here on blood cultures from live calves at the height of the disease has proven negative. In several trials, B. coli was never found, but a micrococcus of the albus variety, referred to before in this paper as probably a saprophyte living in the deeper layers of the skin, developed. This organism was not sufficiently hardy to cover up the colon organism, had it been present. Consequently, we may say that this work confirms that done by Lesage and Delmer.*

Another strong argument against attributing the disease to the activities of the colon organism is the difficulty found in producing it with cultures derived from pathogenic sources. Jensen reported that he was easily able to reproduce the disease in its typical form by feeding cultures to young calves. Most other investigators have had difficulty in doing this or have failed completely. In two attempts to produce the disease in this way, I have fed large quantities of culture (a liter of milk inoculated and incubated for twenty-four hours) with entirely negative results. These experiments can hardly be said to be entirely fair, however, since one of the calves was four days old and the other about a week. The natural immunity of the calf undoubtedly increases by leaps and bounds during the first few days of its life, and the results might have been different, had the infection been fed during the first twenty-four or forty-eight hours after birth. On the other hand, the amount of infection administered was hundreds of times greater than would have been likely under natural conditions.

MODE OF INFECTION

The nature of the disease makes it seem probable that the pathogenic agent enters the body of the calf through the digestive system. Nocard's work served to direct attention from this port to the navel. There is little doubt that Nocard was really dealing with a navel infection. Therein, his disease differs radically

* Since this paper was written, another case bearing upon this question has been observed. A one-day-old calf showed signs of lameness in both knees, and later great prostration. The feces were extremely watery. A blood culture was made, under rigid precautions, and B. coli communior recovered. After death, which occurred three hours later, the organs all showed the same organism. The calf was moribund at the time the culture was obtained. Consequently the significance is in question, but at least it is shown that colon forms may be found in the general circulation for several hours before death finally intervenes. This Lesage and Delmer seem to deny.

from ours. His organism was of the hemorrhagic septicaemia type. The lesions were largely those of septicaemia hemorrhagica. In one case, in attempting to immunize an adult cow to his pasteurella, he reports killing her. The course of the disease was peracute, and all the lesions were those of hemorrhagic septicaemia.

Some of the continental European investigators found diseases resembling calf scours, which originated in the umbilicus; others found umbilical infections of various kinds complicating true scours. The ruptured navel stump of a new-born animal is merely an open wound subject to all the infections and contaminations of any other open wound. In addition, the blood clot which always exists in the umbilical vein makes a first-class medium for the growth of all sorts of organisms, and there is quite ready access to the general circulation by progression up the vein. It is not at all surprising, then, that frequent septicaemias should occur from infection from insanitary surroundings. In most cases these septicaemias present symptoms very much the same as those shown in the acute type of true scours. For this reason, Poels classifies them all in one group. In most localities these umbilical infections are not at all specific, but there is no reason why, in a certain locality where a particular organism is especially abundant, most of these cases should not be alike. This was the condition with which Nocard worked. Rigid navel disinfection is sufficient to cope with this trouble, but the real calf scours is not so easily dealt with.

In the true calf scours, as we see it, the infection does not occur, as a rule at least, through the navel. We do not see the "bloody extravasations around the navel" which Nocard describes. Occasionally simultaneous umbilical infection has occurred with the scours, but in these cases we are more apt to find streptococci or micrococci than colon bacilli in the umbilical vein. No matter how carefully the navel cord may be cared for at birth, calves in a herd where the disease is prevalent will continue to become sick. In the experimental herd during the past winter, five calves, which afterwards became ill with the disease, have been attended at the time of birth. In these cases the chances for umbilical infection were practically eliminated. A week before

calving, each of the pregnant cows was given an antiseptic bath. The vagina was douched frequently in each. When the calf was born, it was caught on a sterile sheet, removed immediately from the dam, and scrubbed clean by hand, using an antiseptic solution. The Whartonian jelly was carefully stripped out of the umbilicus and the entire umbilical stump submerged for fifteen minutes in 1-500 bichloride of mercury solution. It was thoroughly covered and dried with a powder consisting of equal parts of boric acid and zinc oxide. The stall in which each calf was placed had previously been scrubbed and disinfected. The bedding consisted of a fresh bale of clean shavings which had had no chance to become contaminated. Each calf had a stall completely isolated from the others. The feeding utensils were kept scrupulously clean.

Of these five calves which became sick with the disease, two died. The autopsy showed no evidence of umbilical infection in either, but the body fluids were teeming with colon bacilli. In the umbilical vein of one of these animals, colon bacilli were found the same as in all other parts from which cultures were made; in the other calf, the media inoculated from the vein remained sterile — the only place examined that did not show colon organisms. Therefore we can safely regard umbilical infection as an unimportant factor in the production of white scours in this country.

The possible sources of infection may be divided into three groups, namely:

(1) Intra-uterine.

(2) From maternal passages during birth.

(3) From contaminated food stuffs and surroundings after birth.

The concensus of opinion at the present time is in favor of the third group. Under the head of "Natural Infection", Hutyra and Marek (12) have the following to say:

"In infected stables, the virus may be present in the vagina of the mother, on the floor of the stable, or in the litter from which the external genital organs and the udder of the mother become contaminated. The infection results usually during the sucking of the contaminated udder, but has also taken place during par-

turition, and later through contact with litter, from licking the stable walls, etc. Exceptionally, the infection may also take place through the umbilicus (Poels, Jensen). The infected animals may then infect their surroundings with their excrement and urine, thus increasing the possibilities for infection of animals born later. New-born animals fed artificially with boiled milk may become infected from the contaminated hands of the attendants, as well as by the use of dirty feeding vessels."

This statement may be considered as a summary of our most up-to-date knowledge of natural infection of the young animals. For more than half a century, it has been known that the feces are virulent. Anything which they may soil is dangerous to other calves, and a scouring calf generally soils everything within several feet of its limits of movement. The calf pens with the open-work between them, as seen on the best dairy farms, are vicious for this reason. It is little wonder that the disease claims so many victims when once it gets a start. Arrangements for ventilation should be made in other ways, and the new-born calf should have a stall or pen completely isolated from his fellows for the first week or two of life, at least.

Mention is made of the opportunity existing for infection to occur at the time of birth. To speak about this possibility intelligently, it is necessary to have some idea of the flora of the vagina. In nine cases, Mohler and Traum reported a large number of different organisms, some of which they were able to identify, but the majority not. Of these types, probably the majority were only transitory. It can not be doubted that conditions are quite favorable for the development of saprophytic organisms. Warmth and moisture are present, and no bactericidal substances exist there as far as is known. In the human family, Harada (14) has shown that a definite bactericidal power is resident in the vagina and that this is increased as pregnancy advances. This has not been observed in cattle; there seem to be as many bacteria present, both in numbers and varieties, on the 275th day of gestation as upon the 75th.

To determine the frequency of the colon bacillus, swabs were made from the vaginae of apparently normal cows, pregnant for periods varying from three weeks to one day before calving. Of

nineteen cows examined, B. coli communior appeared four times, B. coli communis once, and the other fourteen animals did not show any organisms of the colon group. This seems to relegate this group of organisms to the transitory variety. Due to the soiling of the vulva with the feces, occasionally colon bacilli grow for a time in the vulva and vagina, but they apparently do not find the parts a favorable site for continued multiplication. The fact that they are present at times makes it possible for them to enter the mouth of the calf as it is being expelled. Whether they are virulent or not, even if swallowed, is another question that needs be answered. According to Jensen's teachings, the infection of the calf with even a purely saprophytic strain of the colon bacillus, before any nutrition has been taken to start the digestive processes, may cause this form to attain pathogenicity. As said before, this assertion is warmly disputed by Poels.

The third possible source of infection suggested was within the uterus. This source was early suggested by Nocard (5) long before his work upon the disease in Ireland. The principal reason for this suggestion was the extreme rapidity of the development of the disease in many cases, after birth. Frequently the calf died within the first twenty-four hours. Apparently this time is too short for infection to occur after birth and for the changes leading to death to occur. Therefore, he concluded that the disease must have made considerable progress before the calf was born. So far as can be learned, no effort was made to prove the correctness of this theory by experimental work.

Hutyra and Marek (12) state that the possibility of intrauterine infection was proven by Kitt, who injected three cc. of a washed agar culture of colon bacilli into the jugular vein of a pregnant cow. The calf, which was born eight days later, began immediately to scour profusely. The basing of such a far-reaching conclusion upon a single case seems unwarranted. That the colon organisms which were causing the diarrhea were the progeny of those introduced into the vein of the mother, there seems no way of proving. Such a conclusion, to have any value, should be based upon a number of similar cases. That colon organisms can get from the maternal circulation to the body of the calf in any way seems to be very doubtful. Nocard (2) in one of his

experiments inoculated a cow in the last stages of pregnancy with a culture of his white scour pasteurella. Death was soon brought about, and on autopsy the same organism was found in all the tissues of the mother. All the cultures from the body of the fetus remained sterile. The organisms, although abundant in the blood of the dam, had not passed the placental filter. In repeated instances, we have shown by the immunization of the dam to colon bacterins that not even the colon antibodies are able to penetrate the filter.

About a year ago (1916) work was begun on the meconium of new-born calves, in connection with the general problem of contagious abortion and allied diseases. The observation has been made by Williams (15) that the meconium always contained hair which was of the same color as that upon the exterior of the calf. Sometimes this hair makes up an important part of the total bulk of the meconium. This finding disclosed a fact in connection with the physiology of the fetus hitherto unknown — that the amniotic fluid is constantly being swallowed by the fetus. Since normally the amniotic fluid is always clear, the digestive tract must act as a sort of filter and the hair, shed epithelial cells, and other debris be retained in the intestinal tract. This being true, a bacteriological examination of the meconium should give an idea of the organisms present in the amniotic fluid in case any are present.

The meconia of fifteen new-born calves were examined. Fourteen of these animals were in two different herds — six in the first and eight in the second — and one was an unborn calf obtained at an abattoir. In most cases the material was collected within a half hour after birth; in no case did more than ten hours elapse. After a thorough scrubbing of the anus with an antiseptic solution, the fore finger, disinfected, was introduced into the rectum and a small amount of meconium removed. This was carried to the laboratory in a sterile Petri dish, flamed, and the sticky mass spread open aseptically. Cultures were made from the middle portions into media enriched with sterile bovine blood serum, provisions being made for both aerobes and anaerobes.

In the first herd, a comparatively large one, the loss from calf

scours this year has been almost negligible; probably not more than 3 or 4 per cent. of the calves ever showed any symptoms. The second herd is a small one, in which only six live calves were produced during the year. Of these six, five became sick with the disease. One calf was brought into the herd when one day old and came down with the disease after a short time. One calf was born dead. The abattoir case was that of a calf near to full term which was removed from the uterus of a slaughtered cow on the killing floor. The calf had been scouring badly; the amniotic cavity was filled with diarrheic feces.

The meconia from the six calves in the first herd all proved to be sterile. In the second herd, of the first six calves, one sample of sterile meconium was obtained, and the other five gave growths. Three of these cultures were of colon bacilli, one was a streptococcus, and one was mixed — a streptococcus and a micrococcus. The meconia of the calf born out of the herd but brought in at one day of age, and of the stillborn calf, were sterile. The abattoir specimen gave B. coli.

Table I gives the data in a comprehensive form.

Table I

	Meconium	White Scours	Remarks
First herd	1. Sterile	—	
	2. Sterile	—	
	3. Sterile	—	
	4. Sterile	Slight, recovered.	
	5. Sterile	—	
	6. Sterile	—	
Second herd	1. B. coli	+	Born in herd.
	2. Sterile	+	Born in herd.
	3. B. coli	+	Born in herd.
	4. Strep. and staph.	+	Born in herd.
	5. B. coli	—	Born in herd.
	6. Strep.	Died suddenly without scouring, Septicemic symptoms. B. coli isolated.	Born in herd.
	7. Sterile	+	Born out of herd.
	8. Sterile	.....	Stillbirth.

Since this study is highly interesting and suggestive, further work was planned along similar lines in order to strengthen and broaden its scope. The fact that the meconium is so frequently infected led to the inevitable conclusion that the cavity of the uterus, contrary to generally accepted opinions, though possessing a normal seal of pregnancy, frequently contained infection other than that of the B. abortus Bang. To get some idea of the frequency of this, arrangements were made to go on the killing floor in an abattoir in one of the packing centres to study pregnant uteri of cows slaughtered and to make bacteriological examinations of them.

These uteri, selected indiscriminately, embraced all stages of pregnancy. A plumber's gasoline blow-torch was used for flaming the outer surfaces and knives used for making the incisions. The incisions were made either close to the os uteri internum or at the ends of the horns where it was thought that infection, if present, would be most likely to exist. The fetal membranes were left intact by the stroke of the knife as the uterine wall was incised. A cotton swab, such as used in diphtheria work for throat swabbing, was inserted between the uterine wall and the outer membrane (utero-chorionic space). After rotating the swab a few times to collect mucus and any bacteria that might be present, it was withdrawn and immediately dropped into a tube of sterile bouillon for transportation to the laboratory. Samples of the

fetal fluid were withdrawn, by plunging a sterile pipette through the membranes into one or the other of the cavities and removing three to four cc. in a sterile tube. Further than this, in the beginning, samples of blood were removed from the umbilical veins of the fetuses by flaming, making a longitudinal incision into it, and passing a pipette into the intra-abdominal part of the vein. About two cc. of blood were obtained in each case. This was placed in about 5 cc. of sterile bouillon for transportation. Cultures were made from all samples within twenty-four hours, in bovine serum-enriched media. The results are tabulated in Table II.

TABLE II

Case	Utero-chorionic space	Fetal fluids	Fetal blood	Approximate duration of pregnancy	Remarks
1	B. coli	B. coli	Sterile	5 months	
2	M. albus	M. albus	Sterile	7 months	Exudate present.
3	B. coli	Sterile	Sterile	5 months	
4	B. coli	Sterile	No sample	5 months	
5	Streptococcus	Sterile	Sterile	8½ months	
6	B. coli	B. coli	Sterile	5½ months	
7	B. coli	Sterile	Sterile	6 months	
8	Streptococcus	Streptococcus	No sample	4½ months	
9	Paracolon	M. albus streptococcus.	A few colonies M. albus.	5 months	
10	Streptococcus				Non-pregnant.
11	B. coli	Sterile		4 months	
12	Sterile	Sterile		3 months	
13	Sterile	Sterile		2 months	
14	Near cervix — sterile apex — streptococcus, M. albus.	Sterile		2 months	
15	Near cervix — sterile apex — streptococcus, M. albus.	Sterile		6 months	
	R. horn — M. albus.				
16	L. horn — B. coli.				Non-pregnant.
17	B. coli				Non-pregnant.
	M. albus				
18	M. citreus				Non-pregnant.
19	Sterile				Non-pregnant.
	Near cervix, streptococcus.				
20	Apex, sterile	Sterile		3½ months	

A summary of the data given in Chart II shows that in sixteen pregnant cows the utero-chorionic space gave cultures in fourteen — about 87 per cent.; the fetal fluids gave cultures in five cases — about 33 per cent.; and only one culture was derived from the seven samples of blood. This is regarded as probably a con-

tamination of the pipette with the amniotic fluid, since it contained the same organism as was in the culture from the blood.

These studies bear out quite well the previous results from the meconium. The meconium has been found infected with B. coli, micrococci, and streptococci. So have the fetal fluids. In all cases except Case 9, whenever the fluids have been infected, the utero-chorionic space has been found to harbor the same organisms, but in nine other cases the utero-chorionic space has been found infected and the fluids free. This shows that the space is furnishing the infection which passes through the membranes in the inter-cotyledonary spaces into the fluids. Since no evidences of an inflammatory condition of the membranes are ever noticed, it may be that the necrotic parts of the membranes, which are found nearly always at the ends of both gravid and non-gravid horns, are the open doorways for the passage of the infection. This will admit it only into the allantoic fluid, and it must still penetrate the amnion to reach the amniotic cavity and the fetus. That much resistance is offered to the passage of the infection by the membranes is evident from the fact that the fluids are uninfected in about two-thirds of the cases when infection is present in the utero-chorionic space.

The question now arises: How does the infection reach the utero-chorionic space in a normally sealed uterus? The idea has become quite prevalent, largely due to the teaching of Williams (16) that the seal, which forms soon after an animal becomes pregnant, is impregnable to bacteria. That this is true has never been definitely proven. Of the five non-pregnant uteri examined, four contained infection and one did not. Roughly, this may be said to correspond to the conditions when the seal was present. The proportion of infected organs is the same, and the infecting organisms are the same. No examination of the vaginae was made in these particular cases, but the results in a number of other cases show that a large number of different varieties of bacteria are always present. Why do they not pass the cervix and invade the uterus? No mechanical impediment exists. The conclusion is drawn that the healthy uterus has a bactericidal power towards most varieties. This leaves the question of the permeability or impermeability of the seal still undecided, but reduces

considerably the practical importance of the question. Obviously, one of two things must be true: either the seal is permeable to certain varieties of bacteria, or certain bacteria are able to remain alive in the uterus, after it is sealed, throughout the period of pregnancy. Either the barn is locked after the horse has been stolen, or it is locked previous to the theft, with a lock to which the thief holds a key.

That infection of the pregnant uterus may occur from the blood stream of the dam is regarded as highly improbable. In a non-pregnant animal, the phenomenon of menstruation affords an opportunity for infection to occur from any organism that may be floating in the blood stream, but nothing of this kind occurs normally in a pregnant uterus. The unruptured capillaries of the maternal placenta are almost surely impervious to the passage of bacteria; should these allow the infectious material to pass, it is unlikely that the contiguous fetal capillaries could prove capable of holding it back, and infection of the fetal blood would occur. This does not occur.

These studies throw considerable light on the mode of infection in white scours of calves. The evidence is not complete and conclusive, because of the uncertainty of the actual etiological factor, but it is at least quite suggestive. That scours is sometimes contracted in utero is proven beyond question by the fact that the disease has been found well developed in unborn calves. These observations seem to show that it is quite frequently contracted in utero.

Without making an attempt to enter into the therapeutic side of the question, a few words in conclusion in regard to the practical application of these results are fitting. In the past, rigid disinfection has been the basis for the control of the disease. While these measures are highly necessary to prevent the spread of the infection from the sick to the well calves, still they will not completely control the situation. Calves born with the infection will develop the disease, despite all antiseptic precautions carried to the nth degree. In these cases the solution of the problem lies in immunizing the calf, if possible, before the disease has developed fully. In some cases the infection has progressed to such a state in utero that the immunization can not be brought

about soon enough and the calf will come down with the disease, in spite of treatment.

In the immunization process, two products are sold and used — white scours serum and polyvalent colon bacterin. Both have their indications, but as a rule the serum is by far the better. In acute cases, an early immunity is the thing desired. This is obtained only by the use of the serum. In the more chronic cases, especially in the pneumonia, the bacterin is indicated instead of the serum.

The injection of a colon bacterin into the dam for the purpose of immunizing the calf in utero is a worthless procedure. A number of experiments have shown that the antibodies formed in the blood of the dam as the result of the injection of the bacterin, even when present in immense numbers, have not been transmitted to the blood of the calf in any instance.

Summary

The disease of young calves known as white scours has been recognized for a century and a quarter, and is very destructive. It is a septicemic disease, accompanied by profuse diarrhea as a rule, and frequently brings about the death of the calf within the first few days of life. Associated with the true scours, there are the more chronic conditions — instead of the diarrhea, joint lesions and a form of broncho-pneumonia. The latter frequently brings about death after a long period of unthriftiness. If recovery occurs, the calf is very apt to be stunted and worthless.

The cause of this disease is now generally considered to be the colon bacillus or some of its varieties, and infection is considered as occurring from contaminated surroundings soon after birth. The proof that these conclusions are correct is not at all complete. That the colon bacillus is at least a powerful secondary invader, if not primary, and is at the base of much of the actual damage accomplished, is unquestionable.

A large percentage of calves are born with infected meconium. B. coli and certain cocci are the organisms found. The same organisms are found in the fetal fluids and utero-chorionic space in the sealed uteri of apparently normal cows. The utero-chorionic space is infected first, then the fluids, and, lastly, the meconium. The infection reaches the meconium by the calf's

swallowing the amniotic fluid. The infection probably reaches the utero-chorionic space by passing through the cervix uteri from the vagina before the seal is formed and persisting there throughout pregnancy.

The infection, frequently existing in the intestine of the unborn calf, sometimes produces scouring before birth, but usually induces an acute toxic condition with diarrhea soon after birth. These diarrheic feces are highly virulent to other calves. Ordinary disinfection is insufficient to deal with this disease, because of the number of calves born with the infection contained within them. These calves will develop the disease, despite the most rigid disinfection of their surroundings and care used with their food.

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FORMALIN IN THE TREATMENT OF MASTITIS

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For our purpose we will divide mastitis into two groups particularly as to cause: first, mastitis caused by infection of various forms; second, mastitis due to other causes than infection. It is of the first group that we wish to speak.

In attempting to overcome the infection I had used the injection of boric acid solution, also oxygen gas without satisfactory results. I had tried the injection of equal parts of alcohol and glycerin as recommended by Schmidt of Denmark. This likewise was not satisfactory and in some cases seemed to increase the infection.

In treating diseases of the respiratory and genito-urinary tract we use antiseptics which, given per mouth, are eliminated through these tracts. Why then should we not treat infection of the udder in the same way?

In reporting for the committee on therapeutics at the 1914 meeting of the New York State Veterinary Medical Society, I reported the good results obtained by feeding methylene blue in cases of mild infection of the udder. I also used methylene blue in combination with turpentine which is likewise eliminated through the milk.

At the 1913 meeting of the American Veterinary Medical Association, Dr. Klein gave a paper on "The Therapeutics of Parenchymatous Mastitis," in which he tells of using boric acid per mouth and finding it in the milk in twelve hours. He also speaks of using urotropin and finding formaldehyde in the milk in twenty-four hours.

At the conference last year Dr. Moak in speaking of streptococcic mastitis gave the impression that it was incurable but later in speaking with him I was unable to find any definite line of treatment which he had used. I had already cured some cases of streptococcic mastitis with turpentine and methylene blue but realized that something stronger was needed.

It was then decided to try formalin and the results were so gratifying in the first case that I began a series of experiments with regard to elimination and dosage.

[illegible]

With the idea of avoiding this we begin the change of the diet [illegible] by the addition of milk and cream [illegible] For this purpose we find effects we gradually increase the change until we obtain the effect desired in the case under [illegible]

We have given 20 c. c. twice daily for two weeks and failed to see that it was producing any effect in any way, either by loss of appetite, vomiting or indigestion. This was at the time we [illegible] We have also given 50 c. c. at one time without any effect.

As to the method of administration we have given it undiluted as above. It may also be given in milk or oil. In administering the latter we get the laxative effect of the oil which would be undesirable in certain [illegible]

Dosage: In determining the dosage of this after giving [illegible] we found 1 dram [illegible] to be the minimum dose which would produce [illegible] Then one dram of formalin was given three times daily and found to be the maximum dose which could be given in the milk in twenty-four hours. When 20 c. c. of formalin was given three hours after meals one hour before bedtime it was found in the milk two hours afterward and continued to be present for forty-eight hours. The largest amount was given from the eighteenth to the thirtieth hour.

We believe it advisable to supplement the formalin treatment with purgatives and diuretics as they increase the intercellular fluid in the tissues and hasten the elimination of waste products from the body. It is also well to use the supporting bandage to relieve the weight and tension on the tissues. Then by giving certain foods the necessary fat for the activities would be given in the tissues is materially reduced. When fat for the night time we have found it necessary to prevent the chilling of the tissues. In fat tissues cold water may be used in place of hot with equally good results.

In our work with formalin we have not used the above methods except in two cases where the animal was necessarily changed from

ensilage to dry hay and in these cases salts were given to relieve any constipation which might result.

In giving the results of our work with formalin, we give one from each of the different forms with which we have been experimenting.

Case I. Cow had calved eight months previously and was pregnant about four months. For the past two weeks she had given thick milk and the two posterior quarters were hard and firm. Cultures made from the milk of this cow were sterile from the fore quarters and from the hind quarters showed a pure growth of streptococci. The animal had been treated with home remedies and cow relief.

Gave one-half ounce of formalin and then followed by one dram dose three times daily alternated with one ounce of turpentine twice daily. Eight days later the treatment was discontinued and on the ninth day cultures from the milk of all quarters were sterile. Five months later, after the cow had again freshened, the cultures were again sterile and the animal averaged over one hundred pounds of milk daily for forty days.

Case II. Cow had calved about four months previously and had been giving thick milk for five days. Cultures from the milk gave pure growth of streptococci from one quarter, mixed growth from another, a pure growth of micrococci from the third and a sterile culture from the fourth. Gave one-half ounce formalin followed by one dram three times daily for five days. Cultures made from the milk on the ninth day were sterile.

Case III. Cow had freshened four days before and had retained placenta. Was giving bloody milk from two posterior quarters. Cultures showed *B. coli* and micrococci. Gave twenty-five c. c. of formalin and repeated for three days. On the sixth day the milk was normal.

Case IV. About one year ago cow had shown a small swelling at base of teat. Two weeks previous to our treatment the swelling had suddenly increased in size and at time of treatment was about three inches in diameter. The swelling was punctured with a hypodermic needle and a thin straw-colored fluid escaped. Cultures from this revealed pure growth of streptococci. Cultures

from the milk of that quarter showed no growth. The abscess was opened and packed with iodoform gauze; the wound covered with sterile gauze held in place by adhesive tape. The second day after the abscess was opened the milk from that quarter became thick. Cultures from that quarter showed streptococci which had undoubtedly passed up the milk canal. Gave twenty-five c. c. of formalin and on the third day the milk was normal to all appearances but no cultures were made. Ten days later the milk became thick and cultures showed a growth of streptococci. This time twenty-five c. c. was continued daily for three days and on the fifth day the milk appeared normal and cultures were sterile.

Case V. Cow had been milking hard for a few days and the trouble was gradually increasing. A small amount of exudate was found on end of teat forming a scab. After removing the exudate the external orifice was found to be normal. Further examination showed a small swelling in the teat canal, at the upper end, which was producing stenosis of the teat and causing hard milking. Cultures from the milk of this quarter showed micrococci and *B. coli.* Gave twenty-five c. c. of formalin and repeated in twenty-four hours. Also painted the base of the teat with tr. iodin and had the exudate on the end of the teat softened with warm antiseptic solution before each milking. Four days after the owner reported that the exudate had stopped forming and the milk was flowing more easily. Two weeks later he reported the cow milking very well.

Much credit is due to Dr. Pickens for making and examining the cultures and also to Dr. Hayden for testing the milk for formalin.

ANAESTHESIA AND NARCOSIS

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I

GENERAL ANAESTHESIA

Disadvantages and dangers attending its use.

Surgical anaesthesia is defined in medical literature as a condition of insenibility or loss of sensation of the nerve centers, of the conducting paths of the nerves, or of the peripheral terminations of the same, usually due to artificial impairment by means of anaesthetics. The general term anaesthetics is employed to indicate substances used to produce unconsciousness or loss of feeling sufficiently complete to allow of surgical operations being performed.

The uses of anaesthesia in veterinary medicine are many: such as to produce muscular relaxation, in hernia, prolapse, dislocations, and fractures; to overcome extreme irritability of the central nervous system as in strychnine poisoning, eclampsia, tetanus or other convulsive affections of the nervous system; in obstetrics, to overcome pain and prevent violent straining. In this paper we will deal principally with the producing of insensibility to pain for operations, examinations, etc.

Every one will admit that operations whether on human or animal patients should be performed painlessly whenever possible, but at the same time the safety of the patient must always be the first consideration of the operator for it is of no avail to have performed any operation, however clever, if the patient succumbs to the anaesthetic.

Inhalation anaethesia has never been popular nor held the place that it should hold in veterinary medicine. This result may have been partly due to ignorance on the part of the old time practitioner, but it still exists principally because general anaesthesia has certain disadvantages in veterinary use which have not yet been overcome.

In the first place, it is to be remembered that veterinarians, except in the larger cities, often live many miles apart. For one

to act as an anaesthetist to the other is oft-times impossible, even if the fee considerations did not stand in the way. No anaesthetic is "safe," and there must always be a certain amount of risk taken by everybody concerned when the anaesthetist is an amateur. The surgeon, himself, may be an expert anaesthetist, but there are times during every serious operation when his mind must be fully occupied by the work in hand, and to divide his attention between two processes will probably spell disaster for both. Again, the veterinary anaesthetist needs expert knowledge of the arrangement of the internal organs of his patients, as the digestive system of one species is totally different from that of another. This is a point of the greatest importance when considering how each animal shall be prepared and secured before the anaesthetic is given. For example: the horse, dog, pig and cat, have each one stomach; the ox, sheep, and goat each have four; the camel has three. Some of these animals are intractable and very bulky, yet they have to be operated upon in all kinds of improvised places. They cannot be placed gently on neat operating tables like human patients nor will they remain quiet and refrain from struggling at the mere request of the doctors and nurses. Canine and feline surgery approaches nearer to human surgery in this respect, for it is possible to manipulate dogs and cats in almost any way one wishes, and to operate upon them in any position which is convenient to the surgeon. The use of anaesthetics for these animals has generally attained a higher level of excellence than its use upon any of the other animals with which the veterinarian deals.

For safety's sake there have been devised on correct principles numerous forms of apparatus which allow of an exact graduation in the strength of the anaesthetic inhaled, but these are exceedingly cumbrous, and while they may be used in human hospitals are not available for the veterinarian. The advantage of this principle of measuring for concentrations of vapors is furthermore only relative, for it has been shown that vapor so diluted as to be absolutely safe does not induce anaesthesia within a reasonable length of time; for example 1 per cent. chloroform seems to be absolutely safe, but no surgeon will wait two hours or even one for the anaesthesia.

Under the most favorable conditions we can say that the dangers of anaesthesia are many though they may be caused only in part by the direct action of the anaesthetic. A fatality may occur at any stage of the anaesthesia, from any one of a number of possible causes, each of which may differ exceedingly in the method of onset and the symptoms presented. We may mention the following causes of death from a general anaesthetic.

1. Early in inhalation, a sudden paralysis of the cardiac ganglion may arise due to a reflex action proceeding from some peripheral injury before complete anaesthesia. This stage of incomplete anaesthesia is a dangerous time to operate, especially in the region of the fifth and other important nerves. This danger can be avoided, however, to a large extent by using a local anaesthetic in conjunction with the general, to overcome the reflex induced by the shock.

2. In the stage of rigidity from tetanic fixation of the respiratory muscles, the blood backing up on the venous side may arrest the heart's action.

3. In the stage of incomplete relaxation, death may ensue from paralysis of respiration, or from paralysis of the motor ganglia of the heart.

4. In the paralytic stage, depression of the vital functions by chloroform narcosis and from shock may result fatally.

5. From twelve hours to two weeks after general anaesthesia death may follow a toxemia caused by degenerative changes in the internal organs. This is an important item to be considered and, as yet, very little literature can be found upon it. It is a well know fact that cats when subjected to general anaesthesia are very liable to die. From our observations we believe that this toxemia is the cause of the greater number of deaths in these animals following operations under general anaesthesia. Subsequent to operation in our clinic a number of cats die in from five to eight days, showing, as a rule, no abnormal symptoms until a few hours before death. In this connection a few experiments were carried out to ascertain if possible the cause of death. On autopsy, all of importance which could be found was an acute parenchymatous hepatitis and nephritis. To ascertain, if possible, whether death was caused by the operations, which in these cases were

principally spaying, or by the anaesthesia, we anaesthetized a number of cats for varying lengths of time; no operation was performed on some, and a few slight external incisions were made upon others. We found that those cats, which were kept under chloroform or a mixture of chloroform and ether for thirty minutes or more, had practically the same train of symptoms as those previously operated upon with fatal results. Upon autopsy these animals also showed an acute parenchymatous hepatitis and nephritis. A few cats which did not die at the usual time were destroyed a few days later. These showed on post mortem examination an acute parenchymatous hepatitis and nephritis, but of a lesser degree than those which died. Acetone was found in the urine of the dead cats. To demonstrate whether this was constant, a urinary examination was made before and after anaesthetizing a few selected cases. The day after these animals were under ether for twenty minutes acetone could be found in the urine, though they were free from it before being placed under the influence of the anaesthetic.

Complications.— The most common complications following general anaesthesia are bronchitis and broncho pneumonia. The former may be rather trivial, but it is a very common sequel, especially after a prolonged anaesthesia in the horse. Chloroform pneumonia in the horse and dog usually makes its appearance on or before the third day with the usual symptoms of that disease. Death in strongly marked cases often supervenes after an illness of but twenty-four hours. In milder cases it may require several days to terminate the life of the patient, but in any event death is usually to be expected.

Accidents.— Among the accidents attending general anaesthesia we may mention the sub-mucous edema of the nasal septum in horses. This is usually brought to notice by marked dyspnea, and, when an examination of the nasal passages is made, it is found that they are considerably narrowed, due to this edema. No definite cause has been given for this, but it is the general opinion that it results from the irritant action of the chloroform upon the sensitive nasal mucous membrane. This in our knowledge is an accident affecting only the larger animals, and the only reliable treatment is tracheotomy.

In the course of operations under general anaesthesia, inhalation of food material into the lungs will occasionally take place. This occurs most often in ruminants. If the operation in these animals is begun on a full rumen digestion may be partially arrested, and some gastric tympany induced, which tends to efface the folds of the mucosa covering the oesophageal opening. Due to the struggling of the patient and to compression of the abdomen, some of the contents of the rumen are forced into the oesophagus and carried to the pharynx, which being paralyzed allows the food materials to drop into the larynx and be drawn into the lungs. It may be added that during a prolonged anaesthesia with the animal prostrate on its side, there is liable to be a hypostatic congestion of the lung which lowers its resisting power, and causes it to succumb readily to the inhaled foreign bodies, and the concurrent invasion of the attendant bacteria.

From the foregoing it will be seen that the inhalation anaesthetics are not yet all that is to be desired from the viewpoint of the veterinarian. However, it is not the idea of the writer to condemn their general use but with these defects in mind to bring out the importance of local anaesthesia either alone, or in conjunction with a narcotic or soporific, which we believe can be substituted in a great many instances for general anaesthesia. No one can deny that there are a number of indications in veterinary medicine for the inhalation anaesthetics where any substitution is not possible as yet, but the decision in these cases lies chiefly with the operator and the urgency of the operation.

LOCAL ANAESTHESIA

General advantages with special reference to shock and distribution of sensation.

Advantages.— It has been said that the advantages of operation under local anaesthesia are entirely with the patient. That is true in so far as life is concerned, but the surgeon, too, can often share in the benefits that arise from such an operation, as his aim is to operate always with the minimum production of suffering, and to reduce his mortality by safely tiding the operative case through the many dangers that necessarily threaten. The special advantages offered to the patient are the absence of

the disturbances incident to general anaesthesia which have been heretofore mentioned. In addition to these we may mention the following advantages.

It is unnecessary to starve the patient before-hand. The alimentary canal should, however, be well emptied by a suitable cathartic, though a light nutritious meal may be given at the regular feeding time preceding the operation. It is claimed that all patients stand local anaesthetics better when fed before-hand, and it is a distinct advantage in preventing weakness or shock in debilitated subjects.

There is not the post anaesthetic disturbance to the alimentary canal such as retching, straining and vomiting in animals capable of the act, which is often so trying to both patient and operator, causing pain and jeopardizing the results of the work especially when this has been about the mouth, intestines or abdominal walls. The possibility of dilatation, intestinal paresis, or tympanitis is eliminated. The regular post operative nourishment is not interfered with; this is of great importance in weakened subjects, and permits a more rapid recovery and convalescence from the operative procedure. Many weakened patients may survive the operation under general anaesthesia, but die from exhaustion due to interruption of nutrition as the result of a disturbed alimentary or respiratory tract.

Local anaesthesia seems actually contraindicated in very few conditions among which we may mention those of very young or highly nervous animals. The additional trauma suffered by the tissues, due to the infiltration, might in some cases of low vitality be regarded as a disadvantage, but it is in just such cases of low vitality that general anaesthesia is the more dangerous.

Shock.—This, when severe, is a condition to which general anaesthesia is contraindicated, as the administration of a general anaesthetic does not necessarily relieve the reflexes of the higher centers. When this condition of shock is marked such administration may prove highly dangerous; and it should not be used when the employment of local or regional anaesthesia is possible. Local anaesthetics block the afferent nerve paths and prevent further impressions being recorded at the higher center. It is well to bear in mind also, that in inhalation anaesthesia only a part of

the brain is asleep. Complete anaesthesia of the brain produces suspended animation, or death. It is claimed that the medulla at least is but little affected, and the response of the unanaesthetized portion of the brain is constantly observed in the course of operations. For example, the altered rate and rhythm of the pulse and respiration, the change in the vasomotor tone, as indicated by the fluctuation in the blood pressure, the contraction of muscles, and, under light anaesthesia, the purposeless movements of the body, all show that a large portion of the brain is anaesthetized only partially, if at all. These subconscious phenomena represent the discharge of nervous energy in response to mechanical stimulation, and are vain subsconscious efforts of defense or escape. Therefore, in all operations where the impressions from the field of operation may favor the development of shock, local anaesthesia should be used to physically block the afferent impulses, thus sequestering the brain from harmful impulses. This is also true even when the animal is under the influence of a general anaesthetic. Whenever in the course of an operation large nerve trunks are to be divided, the nerves should be first injected with an anaesthetic solution before division.

Distribution of Sensation.— Before taking up any of the methods or agents used in preventing pain, it would be well to make some note of the distribution of sensation. The skin is the great sensory organ of the body, and to it are distributed most of the sensory nerves, but the distribution of these nerves varies within certain limits. It is provided that the more a part is exposed and the more delicate its organization the more exquisitely contrived and highly developed is the apparatus for its protection. Note the case of the eye, protected by its lids, which act involuntarily for protection, before the will could set them in motion. Note other parts of the body which are involuntarily withdrawn from the first touch of danger before the will can act.

Certain senses are limited almost exclusively to the skin, as tactility, locality, and thermic sense, although with the latter certain mucous surfaces feel both heat and cold.

Subcutaneous cellular tissue and fat have very little sensation. In the subcutaneous fat tissue, and other parts further removed from the surface, are encountered the pacinia corpuscles, which

are visible to the naked eye as little globular-like masses. They are connected with the sensory nerves, and transmit painful impressions; what other functions they possess, if any, is not known.

Between the muscle bundles are numerous small nerves which are quite sensitive to pain, otherwise muscle fiber is almost devoid of sensation.

The periosteum is quite sensitive, acutely so in the inflamed state. Bones receive nerve fibers from the overlying periosteum, but when the periosteum has been anaesthetized or has been removed, the bone is then quite insensitive. Bone marrow is sensitive, this sensitiveness varies greatly in different subjects. It receives nerve fibers from the same source as the bone, and when these have been anaesthetized or destroyed the marrow is then insensitive. The same can be said of perichondrium and cartilage — the perichondrium is sensitive but the cartilage not so.

Tendon-sheaths are sensitive, but tendons and aponeuroses possess very little, if any, sensation. Synovial membranes are quite sensitive. The mucous membrance of all the passages communicating with the surface are quite sensitive, that covering the gums and hard palate much less so than that of the surrounding parts. Some distance from the external openings these parts lose their sensation. The mucous membrane of the oesophagus and trachea are insensitive; the oesophagus, however, has a limited sensibility for heat.

Vessels (arteries and veins), except of the smallest size, are sensitive to pain, and this is so even in parts ordinarily devoid of sensation. Fat has no sensation, but the vessels which course through fatty tissues are quite sensitive. For example, the fatty structure of the omentum has no painful sensation but the larger vessels are quite sensitive, and should not be clamped, ligated or cut without first blocking them.

It is generally accepted that the normal gastro-intestinal canal does possess the sensation of pain, although some writers claim that most of the pain arising within the abdomen comes from the parietal peritoneum and its subserous layer.

NARCOSIS

Advantages.— Narcotics are agents which do not abolish pain to such an extent that they can be termed *"true anaesthetics,"* but they have a numbing effect on the senses of animals so that operations can be performed upon them without evidences of any great pain, and without the extra risk which accompanies any of the inhalation anaesthetics. It is a well known fact that certain species of animals have well marked idiosyncrasies for the effects of certain drugs, and as an illustration one can not take a better example than the susceptibility of the dog to the narcotizing effect of morphine.

Although such drugs are not true anaesthetics, we believe their use is justified under certain circumstances, especially when their use is combined with local or regional anaesthesia. We believe from our work that this method should be substituted for general anaesthesia whenever possible, as it does away with the greater risk of the latter and animals under the influence of these narcotics do not feel any great pain and are easily restrained. Under this head of narcotics we will mention chloral hydrate and cannabis indica as the preferable agents for the horse and ox, and morphine or its mixtures for the dog. Chloretone and paraldehyde may be used on canine laboratory animals where recovery is not desired.

II

Local Anaesthesia

Local Anaesthetics

History, derivation and physiologic action of recommended agents.

History. — The scientific use of local anaesthesia dates back no earlier than 1884, though ice, snow, cold water, and various other expedients were used ineffectively from time immemorial. Halstead in 1884, Corning in 1885, and Reclus and Wall in 1886, were the first to describe in medical literature subdermal application of chemical anaesthetics. In veterinary surgery, local anaesthetics did not come into general use until the late 90's although there is some evidence to show that they were used here and there by some veterinarians as early as 1888. For convenience of description we may divide local anaesthetics into two classes, those which are injected into the tissues and those which are applied externally.

Under the first head, we may mention the following: cocaine, anaesthesin, alypin, tropococain, stovain, chloretone, eucain, novocain, orthoform, holocain, and quinine and urea hydrochloride. These infiltration anaesthetics usually produce a loss of sensation with a local anemia in one of the following ways: pressure on the tissues, lowering the temperature, or through the specific action of the contained drugs.

Under the second head, we may include the following: freezing, or cold applications, essential oils as: cassia, cinnamon, cloves, sassafras; ether spray, ethyl chloride, alcohol, ether, carbolic acid, carbon disulphid, and many others.

With the introduction of cocaine in 1884 by Koller, began the real development of modern local anaesthesia as, while many other substances had been previously tried, none had been found satisfactory. Many substitutes for cocaine have been introduced in recent years, chiefly because of their lesser toxicity. Few of these have become popular, eucain and novocain being the chief survivors. Solutions of quinine have been quite extensively used by some, their advocates claiming that the toxicity is low, and that the anaesthesia produced is of long duration.

The ideal local anaesthetic, in our mind, should possess the following properties:

1. It should be efficient in producing a durable, diffusible, and maximum analgesic effect, with a minimum of local tissue disturbance.
2. It should be non-toxic to the organism when absorbed in doses required to obtain the fullest local effect.
3. It should be benign in its local action on the tissues, non-irritating, non-toxic; it should not permanently injure the cellular protoplasm or interfere with the normal repair of the traumatized tissues.
4. It should be absolutely sterilizable by heat.
5. It should be soluble in water.
6. It should be thoroughly compatible with adrenalin.
7. It should be, for veterinary work, especially, procurable at a reasonable cost.

Since cocaine was the first introduced, and still remains the standard and most universally employed anaesthetic, it will be described most fully. The newer agents, which have been introduced, will be discussed only to show their comparative advantages or disadvantages to this drug.

Cocaine.— This is the chief alkaloid of erythroxylum coca, and is generally used in the form of the hydrochloride. During the early days of its use the methods of manufacture and purification were very imperfect, and led to its admixture with many impurities, giving rise often to serious accidents when used about the eye, and leading often to a misinterpretation of its normal action. At present the synthetic preparation of the drug has been a means of reducing the cost of manufacture, and has led to the discovery of many synergestic drugs which have proven highly valuable. The following tests have been offered as a means of determining its purity.

Maclagan's: Dissolve one grain of cocaine hydrochlorate in two ounces of distilled water, to which add one or 2 drops of ammonia solution; after stirring for a few minutes, if free from amorphous cocaine, cocaine hydrate will separate from the solu-

tion in crystalline form, and settle at the bottom, leaving the supernatant solution clear and free from opalescence, any cloudiness indicating the presence of amorphous cocaine.

Cartier's: Mix one drop of a 2 per cent. solution of permanganate of potassium with a solution of cocaine hydrochlorate. The resulting fluid must assume a red color and remain transparent. To the solution add drop by drop more of the permanganate solution. There should appear a red precipitate of permanganate of cocaine, this should become brown only after heating, and without giving off an odor of bitter almonds. If the addition of one drop of the permanganate solution produces a brown color or brown precipitate or when upon heating the mixture there is produced an odor of bitter almonds, the preparation is impure and unfit for use.

External action: There is no action upon the unbroken skin because the drug is not absorbed, but if it is mixed with some substance that is absorbed, or injected hypodermically, or applied upon mucous membranes, it produces complete local anaesthesia by paralyzing the sensory nerve endings. The part soon becomes pale on account of constriction of the blood vessels, but this is quickly followed by congestion. Very large doses also paralyze the motor nerves. Anaesthesia take place in from five to ten minutes, and lasts twenty to thirty minutes.

Internally: Digestive tract. Taken internally the gastric mucosa is numbed and the sensation of hunger lessened. Small doses are said to increase peristalsis and large ones decrease. On account of its local anaesthetic action, cocaine is frequently used to control or stop vomiting. Very large doses lead to paralysis of the bowels and constipation.

Nervous system: Cerebrum. Moderate doses strongly stimulate the higher parts of the brain, and in the human family cause a sense of calm and happiness. This is shown in animals by increased normal movements. They have greater endurance, and hunger is diminished. This greater physical energy renders possible the performance of long exhausting muscular feats. It is probably for this reason that cocaine is used as a stimulant in race horses. Large doses lead to depression, loss of co-ordination, narcosis, and cerebral convulsions. The sensory nerves are para-

lyzed and the motor nerves depressed by toxic doses. Symptoms of poisoning by cocaine are quite variable but usually consist of excitement followed by delirium and finally depression, stupor, rapid pulse, hurried respirations, elevation of temperature, dilation of pupils, and convulsions.

Novocain.— With the introduction of this agent the efforts of the investigators of synthetic anaesthetics have been crowned with a marked degree of success, as it is an effective agent, absolutely non-irritant, and of low toxicity. The absence of irritating qualities in novocain is most marked even when applied in powder form or concentrated solution to sensitive wounds in the most delicate tissue, and on such surfaces as the cornea.

It is soluble in water 1 to 1, and in alcohol in 1 to 30; it melts at 156 degrees C. and can be heated to 130 degrees C. without decomposition. Its solutions possess slight antiseptic properties, are capable of repeated boilings without apparently affecting their strength, and may be kept for long periods of time (several months), without apparently suffering any change in their action, a quality not possessed by any of the other anaesthetic agents. Like other agents of this group, its solutions are precipitated by alkalies and alkaline carbonates, with the exception in favor of novocain that it is not precipitated, or its solutions rendered turbid, by sodium bicarbonate.

The physiologic investigation of novocain shows that it produces no mydriasis, no disturbances of accommodation, and no increase in intra ocular pressure. The effect of moderately large doses upon the general system, when given by either the intravenous or subcutaneous avenue, shows almost no perceptible change upon the circulation or respiration. Numerous investigators give its relative toxicity as one-fifth to one-seventh of that of cocaine, while studies made to show its relative activity seem to show 1.25 per cent. solution in anaesthetic activity, to 1 per cent. solution of cocaine. On the other hand, this agent when combined with adrenalin possesses strength equal to that of cocaine and though slightly slower in action, often yields an anaesthesia of longer duration.

Injected within the tissues, even in strong solution, novocain exerts but little or no influence upon the vasomotors of the part.

Its injections are without pain, and seem to be absolutely free from all irritation. No after pain, inflammation, hyperemia, or induration has been observed to follow its action.

Quinine and Urea Hydrochloride.— This occurs as white, odorless, bitter crystals, or white powder, soluble in one part of water, and freely soluble in alcohol. It contains about 70 per cent. of the quinine alkaloid. Its anaesthetic action was discovered by its having been used hypodermically in malaria. The area of injection remained free from sensation for several days. Compared with cocaine or novocain its local anaesthetic effect is not as rapidly obtained. This is especially true of its topical application to mucous membranes. On the other hand, when anaesthesia is obtained it is of very much longer duration, the after pain in some operations being thus avoided. The local anaesthetic effect is not only slower in its appearance, five to fifteen minutes, but is less diffused. Quinine acts as a vasodilator, and favors capillary oozing. It produces a secondary indurative reaction in the tissues, due to a fibrinous exudate, which appears a few minutes after injection, and in a general way is dependent upon the concentration of the solution.

Eucain.— This is a synthetic compound closely allied to cocaine. Its chief advantages over cocaine are that it is less toxic, solutions are more stable, and it may be boiled (sterilized). However it has no constricting action on the vessels, and is more liable than cocaine to cause sloughing.

Holocain is a coal tar product which has been introduced as an anaesthetic for operations upon the eye. It paralyzes the sensory nerves of the cornea more powerfully than cocaine, and does not produce any necrosis. No toxic symptoms have been produced and the drug is not decomposed by boiling. It has no effect upon the bloodvessels. It is used in from 0.5 per cent. to 1 per cent. solution. The anaesthesia begins in about half a minute, and lasts for five to ten minutes.

Tropacocain is obtained from the small Java coca leaves. It resembles cocain in its actions, but is much less toxic and does not constrict the vessels when applied to mucous membranes. Its only advantage over cocaine is that it is less toxic.

Orthoform has the same action on the sensory nerves as cocaine, is almost completely insoluble, consequently is slowly absorbed and is excreted so rapidly that a toxic effect is almost impossible. This factor is of advantage when large sores are to be dusted repeatedly. On the other hand, when made soluble artificially it is no less toxic than cocaine, and has no advantages. It is mostly used in human practice as an anaesthetic dressing, occasionally combined with caustics to deaden the pain of the latter.

Anaesthein resembles orthoform, and has been recommended as a substitute, being stronger and less irritant. There is, however, more danger of systemic poisoning. Its hydrochloride is soluble, and may be used for subcutaneous infiltration anesthesia.

Alypin is a coal tar preparation. It is equally active to, but less toxic than cocaine, causes no mydriasis, or vaso-constriction, but on the contrary, dilatation of the vessels. It resists boiling for five to ten minutes. It is employed the same as cocaine, but is liable to cause more swelling.

Stovain is another synthetic compound; the hydrochloride of an ester, of benzoic acid. It is freely soluble in water. It is about equal to cocaine as an anaesthetic but dilates the blood vessels and is less toxic. It can be used for the same purposes as cocaine. It has been recommended for intraspinal injection (sublumbar anaesthesia). Gangrene may result at the point of injection.

In comparing cocaine and its substitutes, we would say the most valuable features of cocaine are its strong, prompt and quite certain action. Being a vasoconstrictor, it lessens hemorrhage, may be of service as a mydriatic, causes no local irritation and acts on sound mucous membrane. The substitutes improve on the original or older drug mainly by their lessened toxicity, non-injury of the cornea, greater stability, sterilizability and the possession of antiseptic properties. Novocain has given the best results, but for certain purposes is not superior to freshly prepared solutions of cocaine in careful and experienced hands. Alypin is less toxic and seems to produce fine results, but may be followed by swelling for some days.

General Principles

Including choice of a solution, outfit, use of a constrictor and methods employed.

It should be first clearly understood that the artificial anaesthesia of any given tissue or organ of the body is entirely dependent for its production upon the suppression of all sensorial (irritant) impressions made upon that region, through the agency of the nervous system. This suppression can be effected by:

1. Paralyzing the peripheral nerve endings, or terminal organs of sensation, as in the papillary layer of the skin, or

2. By "blocking", or obstructing, the path of all sensorial impressions in the nerve trunks, that connect the field of operation with the sensorium.

Choice of a Solution.— Solution No. 1 is the one recommended for general use, and, in the majority of cases, will be found amply sufficient for all purposes. It is sufficient for skin wheals, and owing to the mild toxicity of the novocain can be used for massive infiltration of the deeper parts. It can be used for blocking small, or even medium sized nerves, but for large nerves we would advise either solution No. 2 or No. 3.

Solution No. 1

Novocain — 0.25 or 0.5 parts.
Normal salt solution (0.5%) — 100 parts.

Solution No. 2

Novocain — 1, 2, or 2¼ parts.
Normal salt solution — 100 parts.

Solution No. 3

Cocaine hydrochloride — 2 parts.
Normal salt solution — 100 parts.

The last two solutions named are intended for use in the more sensitive parts and for endoneural and perineural injections of the larger nerve trunks.

The addition of adrenalin to these anesthetizing solutions is an important step. It is a powerful vasoconstrictor, and in a dilution

of 1 to 1,000,000 will produce a local ischemia. It takes the place in a large measure of the physical aids, cold and pressure, and by hindering absorption acts as a valuable safe-guard against poisoning from the local anaesthetic. It can be added to the solutions drop by drop in the commercial strength (1–1,000), or may be prepared with the anaesthetizing drug in tablet form. Not over 10 drops to a 3 ounce mixture, or 20 drops to a 4 or 6 ounce mixture, should be used for fear of producing unpleasant symptoms.

The idea of using other than watery solution of the anaesthetics (as in oils), has been advocated at various times, and on purely theoretical grounds might seem to have some claims. Practical tests have shown this method to be unsatisfactory, and to have many disadvantages. Oily solutions diffuse very poorly, exert a weaker anaesthetic influence, and frequently prove irritating to the tissues.

As stated above, novocain solutions resist boiling, and may be sterilized in this way. Solutions of cocaine are more or less unstable chemically, and do not stand repeated or prolonged boilings. It is better to have prepared solutions for constant and definite results although they may be boiled once, immediately before using, without injury. Another method of sterilization is to dissolve a definite amount of cocaine in alcohol, allow the alcohol to evaporate, and then dissolve the precipitate in sterile water or salt solution.

Outfit.— The outfit necessary for the application of local anaesthesia need not be complicated. All that is necessary is to have a suitable supply of syringes, preferably glass, with telescope, interchangeable needles. For massive infiltration, where the use of large quantities of solution are necessary, a large infiltration syringe will be found serviceable, as it permits of the easy infiltration of large areas within a few minutes. For average use the size of the syringe will be from 2 to 10 c. c. capacity, varying with the work required. The size of the needle should be as small as practical, because the larger the needle, the harder it is to insert and the greater the pain when introduced. They should be sharp as this facilitates their introduction thereby lessening pain. They should withstand boiling, and be otherwise surgically satisfactory.

The Constrictor.— It is a well known fact that the action of any of the local anaesthetics can be prolonged when the circulation of the part is impeded. If the part is first rendered ischemic, the diluting and neutralizing influence of the blood is removed, allowing the drug to act more exclusively upon the tissue cells and their nerve endings. This also permits of an indefinite prolongation of the anaesthesia, and by this prolongation, the toxic activity of these drugs is greatly reduced so that doses which may have been regarded as dangerous, used by other methods, can often be safely employed in connection with a constrictor. This constriction may be brought about either by the addition of adrenalin called the " therapeutic constrictor," to the solution as mentioned before, or by means of mechanical devices. A mechanical constrictor should be several inches in width and always carefully applied over well padded parts.

Methods.— The methods practiced in local anaesthesia will be considered under the following heads (1) surface anaesthesia, (2) infiltration, (3) regional.

Surface application. Cocaine or other analgesic drugs applied to mucous membranes by means of a spray or swab will produce loss of sensibility. Its use in this way is confined, for the most part, to special work, such as in the larynx or eye. Very strong solutions were formerly used, but the tendency now is to reduce the strength. With the weaker solution, anaesthesia appears more slowly, but in time is just as thorough. The operator should be in no hurry, but should wait from ten to twenty minutes before beginning any step likely to cause pain.

Infiltration. By this we mean an artificial edema produced by injecting fluid into the tissues. This injection must be intradermal, and not subcutaneous; it should develop a distinct wheal, which stands up from the surrounding surface like an urticarial wheal. The first anaesthetic point should be regarded as a " station " from which the anaesthesia is distributed in the desired direction continuously, in an intradermal line, or a long needle on a large syringe can be advanced through the " station " to subcutaneous or deeper parts, and paraneural or other injections made as desired. Whenever the needle is reintroduced it should always be done just within the margin of the last injection;

otherwise each additional needle prick will be felt. The injection should not be too rapidly done, as the sudden distension of the tissues may cause pain, or possibly rupture of some delicate part.

While it is generally advisable to precede any incision by an intradermal infiltration along the proposed line, this is not invariably necessary, as massive infiltration of the subcutaneous tissues reaches the nerves in their course to the skin.

The regional method. This aims to reach the nerves on the proximal side from the field of operation. The anaesthetizing fluid is distributed about a nerve trunk so that it is bathed in it (perineural), or is injected directly into its substance (endoneural). In perineural injections, the time required to produce anaesthesia and the thoroughness of the anaesthesia naturally depend upon the size of the nerve trunk and the strength of the solution. Strong solutions No. 2 and No. 3, being advised for the larger nerves, and No 1 for some of the smaller. In endoneural injection the needle is thrust directly into the nerve, and the fluid injected until the nerve is swollen and edematous. The needle should point centrally, as this prevents traction, and causes less pain, although there is ordinarily little manifestation of pain when the needle is introduced. The conductivity of the nerve to all impulses is immediately blocked. Endoneural and perineural injections have their greatest field of application in the surgery of the extremities, the latter being the most practical as the former is of service only when the nerve trunk is exposed by dissection.

CLINICAL APPLICATION

Skin and Subcutem

1. Infiltration. The most satisfactory method of anaesthetizing the skin is by the production of a wheal with a weak solution of a local anaesthetic as mentioned before. These infiltrations of the skin and subcutem have a great and unique value in all operations upon cutaneous and subcutaneous structures supplied by cutaneous nerves (for example in the removal of saddle and harness galls, tumors, the drainage of certain abscesses, and in the treatment of wounds). A definite part can be anaesthetized by producing a wheal completely around the area, thus blocking the nerves at a distance. When the operator desires to make an

incision into the underlying tissues, all that is necessary is to first narcotize the skin, and then make injections to the depth of the incision. If the operation is made upon a tumor or like elevation a few deep injections made directly underneath the operative area will cut off all sense of feeling.

The value of the addition of adrenalin to the anaesthetic as a constrictor is clearly seen in skin work. To illustrate this we offer the following:

Areas on the necks of several horses were shaved, disinfected, and a simple aqueous solution of cocaine injected. When anaesthesia was complete, usually at the end of five minutes, incisions were made, and the amount of bleeding noted. Near the same incisions injections were made with the same solutions, except for the addition of a few drops of 1–1,000 adrenalin. When cut, this tissue was blanched and white and did not show nearly as large an amount of hemorrhage as the first mentioned incisions.

The following cases will serve to illustrate the use of skin and subcutaneous infiltration.

Case 1. Adult collie, with a tumor of the left posterior mammary gland, about the size of a small apple. A skin wheal of novocain 0.5 per cent. was raised about the operative area, and several injections were made into the deeper structures underneath the tumor. The entire removal of the tumor, and closure of the skin wound, was performed without any manifestation of pain.

Case 2. Adult hound: operation, taking up the femoral vein. A skin wheal was raised with 0.5 per cent. novocain along the line of proposed incision, and about 2 c.c. of the same solution injected into the structures about the vein through this skin wheal without removing the needle. Operation performed without the least manifestation of feeling or pain.

2. Freezing. The usual means by which cold is employed in surgery for its local anaesthetic effect is by the use of various gases or liquids of low boiling point, which are usually projected in the form of a spray upon the skin, their rapid evaporation producing an intense cold which freezes the part. The rapidity and intensity of action of these agents, depend, aside from their power to abstract heat, upon the vascularity of the part, and the duration of their action is influenced by the same factors. In

highly vascular tissues their freezing action is less marked, and of shorter duration, than when the opposite condition exists. In parts where the circulation can be controlled a more intense action is obtained, which is also of much longer duration, and this action is further increased if the part is first rendered ischmic. When used under these conditions, however, great care is necessary to avoid permanent injury to the parts resulting from coagulation of the blood in the superficial vessels, terminating in localized gangrene.

When it is desired to obtain the most intense action of the cold, in addition to the exsanguination of the part above mentioned, it is well to remove all fat from the skin by the action of ether or benzine, though on the other hand it may be desirable to protect particularly tender tissues from a too violent action of the agent by first smearing them lightly with vaseline. These freezing sprays, of which ethyl chloride contained in a Gebauer tube stands first, are best suited to superficial minor operations which do not involve much more than an incision. They are particularly indicated where it is not advisable to infiltrate an inflamed or infected area with a local anaesthetic, and their special claim for usefulness is in the time saved. They are useful in opening boils or superficially situated abscesses, and in the removal of foreign bodies, such as splinters, etc., from beneath the skin. When skillfully used as a continuous spray they are also efficient in doing some major work. For example, by their assistance the writer was able to castrate a full grown dog without any serious pain.

Its clinical application may be illustrated as follows:

Case 1. Animal was an aged, rather nervous bay mare, on which it was desired to make an incision through the skin of the neck, down to the muscle fascia. The area was shaved, and disinfected with alcohol. Ethyl chloride spray was applied, until a white line was noticed. The anaesthesia was complete in two minutes, and the incision was made without the least manifestation of pain.

Case 2. Animal an adult male hound, for castration, which was placed on operating table, and scrotum cleaned and disinfected. A white line of anaesthesia was completed with ethyl chloride over both testicles, along the proposed line of incision,

and the cuts made. Similar anaesthesia was produced after the cord had been drawn out. In crushing the cord with an emasculator there was slight pain, but of no such degree as that evidenced when the operation is performed without any anaesthesia.

Mucous Membrane and Delicate Tissues. Cocaine, or other analgesic drugs, applied to mucous membranes by means of a spray or swab will produce loss of sensibility. In our experience, cocaine in from 5 to 10 per cent. solution is the most desirable preparation, although other drugs, as novocain, alypin, etc., may be substituted. This method of application is best brought into use in the mucous membranes of the mouth, larynx, in the region of the rectum, and in other parts where the skin is thin, tender, and denuded of hair. The technic consists simply of applying the solution with a swab or spray to the desired area, and allowing enough time to elapse (ten minutes will suffice in most cases), before attempting to operate. Areas underneath mucous membrane may be infiltrated with a weak solution of novocain, injected directly beneath the anaesthetized area, and surgical work done without pain. This surface method of application of cocaine to mucous membranes is of special value in the roaring operation performed under local anaesthesia with the patient in the standing position. The following cases will illustrate its application.

Case 1. Bay gelding for roaring operation. Line of incision on mid line infiltrated with 0.5 per cent. novocain solution and a painless cut made into the larynx. Both ventricles were swabbed out with dry cotton to remove excess of secretions, and 5 per cent. solution of cocaine plus a few drops of adrenalin injected into each ventricle, with a long nozzled syringe. After waiting five minutes the mucous membranes of the ventricles was removed with the burr method, without pain or disturbance to the animal, except that caused by the mechanical manipulation.

Case 2. Applied with a swab for three minutes a 10 per cent. aqueous solution of cocaine to inguinal region of an aged collie bitch. Allowed ten minutes to elapse before incising the skin, which was done without the least sign of pain being evinced.

Lymphatic Glands. Isolated groups of superficial diseased glands, when well defined, can be easily removed after infiltration of the surrounding parts. The submaxillary, pre-scapular and

precrural glands are quite accessible for extirpation under local infiltration and no special technic is required. As an example:

Case 1. Grade Jersey cow, with enlarged tubercular precrural gland. The skin and tissues around and underneath the gland were infiltrated with 0.5 per cent. solution of novocain, and no pain was evinced on surgical removal of the gland.

Joints and Synovial Sac. All articular tissues contained within the synovial sacs can be anaesthetized by passing a needle into the sac, and filling it to a point of moderate distention with a solution of 0.5 per cent. novocain, or a slightly lesser quantity of a stronger solution. This is allowed to remain for about five minutes, when it can be withdrawn, or it will escape from the incision, if any is made.

The Eye. The application of local anaesthetics to the eye is so well known that little will be said about it, with the exception of a brief discussion of the drugs especially recommended for this purpose, this being included for the purpose of bringing out their comparative advantages and disadvantages. Cocaine hydrochlorate, usually employed in 2 or 4 per cent. solution. A 10 per cent. solution has been occasionally advised, but is very liable to produce bad results. This drug causes drying and roughening of the corneal epithelium, which may be partly avoided by keeping the lids closed after each instillation. In wounds of the cornea, cocaine should not be too freely used, as it may interfere with the healing. Gelatin discs impregnated with cocaine have been recommended by some, but the general opinion is that they have no advantage over a solution of the drug. That cocaine is of marked therapeutic value as a local anaesthetic in eye work may be proven by the following.

Case 1. Brindle bull dog, suffering from ophthalmia and photophobia, due to the irritation produced by the membrane nictitans of both eyes, which were enlarged, protruded and apparently paralyzed. A 4 per cent. solution of cocaine was instilled into both eyes, and after waiting five minutes the operation of excising each membrana nictitans was painlessly performed.

Case 2. Brown saddle mare. Foreign body on cornea. Animal was apparently in great pain and unable to keep its swollen and

tender lids open. A 4 per cent. solution was instilled in the eye, and after waiting four minutes, care being taken not to touch the eye lashes, the foreign body, a piece of straw, was removed with ease.

Holocain. When used in 1 per cent. solution, this causes anaesthesia in from fifteen seconds to one minute, the condition lasting about ten minutes. It is claimed that this drug does not enlarge the pupil, affect accommodation nor increase intraocular tension; also that its solutions have some bactericidal properties.

Case 1. Horse. One per cent. solution instilled into the right eye. Anaesthesia was marked at the end of one minute, and remained for about fifteen minutes.

Case 2. Dog. One per cent. solution instilled into left eye. Anaesthesia was present at the end of forty seconds, and remained for eight minutes.

Dionin. A morphine derivative, which produces within a few seconds after its application, smarting, burning, stinging, and marked edema of the conjunctiva. Within twenty minutes this reaction subsides, and analgesia appears, which lasts from three to twenty-four hours. Dionin though an analgesic, is not a true anaesthetic, and in a sense does not belong to this group, but the writer has used it in solution of from 1 to 5 per cent. in a number of cases of painful keratitis in dogs following distemper, with apparently good results.

Quinine and Urea Hydrochloride. This is sometimes used to produce a prolonged analgesia in certain ailments of the eye where pain and photophobia are present for some time. The powdered drug may be introduced directly into the conjunctival sac at the inner canthus, after first washing the parts with sterile water. Within a short time it promotes a flow of lachrymal fluid, and an anaesthesia is present usually at the end of twenty minutes, and lasts for several days.

Case 1. Powdered three-quarter grain of quinine urea hydrochloride in the right eye of a large setter dog, testing both eyes for sensation at the time. A marked increase in the lachrymal fluid was present in three minutes, and anaesthesia was present within one-half hour. This analgesia persisted for over thirty-six hours when the animal passed from notice.

Stovain and Alypin. These both produce an anaesthesia, but are liable to cause smarting, burning, lacrymation, etc. In the writer's opinion they have no advantages over the above mentioned drugs, and should not be used in eye work.

Enucleation of the Eye Ball. This is easily performed under the technic of ordinary infiltration. Almost perfect results may be obtained by painting the cornea with a 4 per cent solution of cocaine, and making four infiltrations above, below and on either side of the orbit, deep into the orbital cavity. It may be necessary in some cases to infiltrate the cornea at the border with a 0.5 per cent. solution of novocain. The following case may be taken as an example.

Case 1. Black medium sized spaniel for enucleation of the eye ball, following injury. The cornea and conjunctiva were swabbed with a 4 per cent solution of cocaine, and about one-half c. c. of 0.5 per cent of novocain injected above, below, and on either side of the eye ball, down into the orbital cavity. The eye was removed by the regular technic, without manifestation of pain.

The Ear. Besides the regular infiltration methods to be used on the base of the ear, subcutaneous tissue, etc., we wish to make special mention of the surface method in the external auditory canal, particularly in ulcerative otitis in dogs. In this condition, as proven by numerous observations in our daily work, the application to the denuded surfaces of 1 per cent cocaine or 1 per cent. novocain solutions aids the effect of any curative agent, as the marked relief from pain lessens the continual shaking of the head with its attendant bad results, so common in these animals. The above mentioned solutions can be swabbed directly on the ulcerated areas, or injected into the ear with a small syringe, and worked into its depths by the fingers of the operator from the outside.

Head and Neck. Although delicate operations upon the head are not as common in veterinary work as in human practice, local anaesthesia in this region has a wide field of usefulness. Operations upon the skin and underlying bones are easily performed by simple infiltration of the area around the proposed line of incision, together with the infiltration of the periosteum. This allows the sinuses to be opened, and their depth explored. Wounds, cysts, and benign tumors may be removed by creating a wall of anaesthesia around them.

The definite supply of the fifth nerve, with access to its branches at their exit at the infra orbital foramen, makes the anaesthetization of its distribution an easy matter. Plastic operations about the outer ear, mouth, nose and eye lids are easily executed with the aid of simple infiltration. Major operations about the neck and shoulders were performed with little evidence of pain by infiltration of the skin along the proposed line of incision, and massive infiltration of the underlying tissues, together with proper restraint. The following will serve as examples of local anaesthesia in these regions.

Case 1. Grade Holstein cow, age six years, pyemia of the frontal sinus. Solution of 1 per cent. novocain, with a few drops of adrenalin, was injected into the skin and periosteum over the proposed area for trephining. Absolutely no pain was shown when the skin was cut, or when the circular piece of bone was removed with trephine.

Case 2. Aged bay gelding, for poll evil operation. Animal was given one ounce of chloral hydrate, and forty minutes later placed upon operating table. Proposed line of incision in the skin injected with 0.5 per cent. solution of novocain, and the deeper structures with a 1 per cent. solution of quinine and urea hydrochloride. No pain was shown during the operation, except at one time when the incision was carried outside the infiltration area in order to allow drainage. The patient made a good recovery, and showed much less post operative distress than is shown by similar cases when operated upon under chloroform anaesthesia.

Case 3. Four horses were injected with 1 per cent. cocaine, over both fifth nerves where they emerge from the intra-orbital foramen. Anaesthesia was complete along their distribution in ten minutes, giving an anaesthesia of the nasal structures, and in the lateral portions of the upper labium. This loss of sensation was complete enough to allow of neurotomy of this nerve, as well as operations upon the above mentioned structures. By simultaneously injecting the fifth pair of nerves and the anterior palatine nerve pain in the median portion of the upper labium could be controlled.

The Thorax. This is not so important a field for local anesthesia, as the number of operations in this region are com-

paratively few in veterinary practice. The nerves of the thorax are principally the intercostals and if desired a neuro regional anaesthesia may be procured below the point of their injection. These nerves descend in the intercostal spaces, at first between the intercostal muscles, and lower down chiefly between the pleura and internal intercostal muscle. In the anterior spaces the nerve lies along the posterior border of the rib, in front of the intercostal artery; further back it lies behind the border of the rib, with the artery in front of it. The intercostal nerves give off three series of cutaneous nerves, the upper ones emerging through the serratus magnus, and the external intercostals, about parallel with the digitations of the external oblique. For a wide distribution of anaesthesia it is best to block these nerves above the lateral branches, although they can be reached at any point in their course.

To do this, a line of cutaneous anaesthesia is drawn down the back, over the proposed line of injection. A fine needle is passed obliquely from below and behind, up to the interval between the ribs, injecting as it is advanced, until it strikes the bone; it is then pushed upward and inward for about 1 cm. further, into the intercostal space above, and this freely infiltrated. This procedure is similarly carried out for as many spaces as indicated, taking in two or three spaces anterior and posterior to the proposed field, as the lateral branches of these nerves freely overlap. This technic has been worked out on dogs with fairly good success. No experiments were made upon the larger animals, as no call was given for its application.

The Abdomen. With reference to operations involving only the abdominal parietes, without invasion of the peritoneal cavity, the ordinary technique of the infiltration method applies. The parietal peritoneum is intensely sensitive to division or traction but not to light pressure, heat or cold. The abdominal viscera, on the other hand, apparently possess no marked sense of pain. For the mesentery these rules are not absolute, but it is considered by most writers to be insensitive. Therefore, before incising the parietal peritoneum, or its subserous connective tissue, it should be widely infiltrated to allow retraction, and in handling viscera, dragging on the mesentery or parietal attachments should be

avoided. Incising, suturing, burning, or crushing any part of the gastro-intestinal tract, does not cause pain so long as there is no dragging on the parietal peritoneum, directly or through the mesentery. Operations requiring the opening of the abdominal cavity are not performed frequently in the larger animals as the horse and cow, but are of frequent occurrence in the dog and cat. For the dog we have in morphine such an ideal agent that it is rarely necessary to supplement it. In some cases, however, it may be desired to perform an operation absolutely without pain, and the combination of this with local anaesthesia gives an ideal result. In these cases all that is necessary is to infiltrate the parietal peritoneum and block the folds of the messentery or any attachments of the parietal peritoneum, with a local anaesthetic before injuring or placing traction upon these tissues. To illustrate this we may cite the following cases:

Case 1. Patient was an adult black and white hound, for ovaro-hysterectomy. She was given one and one-half grains of morphine subcutem and twenty-five minutes later placed upon the operating table and the usual antiseptic precautions for this operation taken. The abdominal wall, down to and including the peritoneum, was infiltrated with 0.5 per cent. solution of novocain and incised; the horns of the uterus were located, and the ovarian ligament on both sides between the ovary and parietal peritoneum infiltrated with the same solution. The ovaries and the body of the uterus were then removed by compression and torsion with Ferguson's forceps, and the abdominal incision closed with two rows of sutures. At no time during the operation was there the least pain manifested, and the animal made an uneventful recovery.

Extremities. The most fruitful fields for the application of neuro regional anaesthesia are found in the extremities. This is true because the large nerve trunk can be readily reached, and also because each nerve supplies extensive areas. The practical value of this method on the extremities is evidently very great, due to the fact that a very large percentage of surgical operations are performed upon these structures. In equine surgery the first nerve of importance that can be readily anaesthetized is the median. Numerous attempts have been made by the writer to

obtain a narcosis of the entire limb by injecting the brachial plexus, but as yet, results are not satisfactory. The following results will be cited to show that, by careful injections, a surgeon may obtain either a complete or a partial anaesthesia of the entire limb, according to his wish. Only a few demonstration cases are cited under each head but the general conclusions reached are further supported by observations made upon numerous clinical and experimental animals.

1. *Thoracic Limb.*

Median Nerve. The most accessible point for injection is on the inner surface of the limb just behind the radius, and immediately below the humero-radial articulation. Both cocaine and novocain solutions have been used, and the results are quite uniform. Perineural infiltrations about the median nerve produce a greatly diminished sensibility over the entire median side of the limb. This narcotization is not complete, however, due to the ramifications of the cutaneous branch of the above nerve, but if skin infiltration is used at the point of operation very little difficulty will be experienced. Operative anaesthesia is effective however, at the point of injection over the trunk of the nerve. Median anaesthesia is practical for the neurotomy of the above mentioned nerve, as well as for the diagnosis and relief of lameness due to certain diseases of the anterior limb.

Bay gelding. Right anterior limb, injected with 5 c.c. of a 1.5 per cent. solution of cocaine, over median nerve.

Black mare. Left anterior limb, injected same area with the above solution.

Bay Gelding. Left anterior limb, injected with 8 c.c. of 1.5 per cent. novocain solution over median nerve.

Black Mare. Right anterior limb, injected with the same per cent. novocain solution.

Results in all cases were evident at the end of ten minutes, and lasted from twenty-five to forty minutes. The entire median side of the limb showed a marked loss of sensation, but not complete operative anaesthesia.

Ulnar Nerve. The injections for this nerve are made above and behind the carpus, at a point 10 cm. above the pisiform bone, and in a groove between the middle and external flexors of the

carpus. A single injection upon the ulnar nerve gives an anaesthesia of the posterior carpal region, and is of little practical value except for neurotomy of this nerve. The following cases will illustrate:

Bay Mare. Right anterior limb, injected over ulnar nerve with 5 c.c. of 0.5 per cent. solution of novocain.

Bay Gelding. Right anterior limb, injected one grain cocaine in 5 c.c. sterile water over ulnar nerve.

Black Mare. Left anterior limb, injected one grain cocaine in 8 c.c. of sterile water over ulnar nerve.

Results in all cases were evident in ten minutes and lasted twenty minutes. Each gave the above mentioned area of anaesthesia over the posterior region of the carpus.

Median and Ulnar Nerves. The simultaneous injection of these nerves gives a complete loss of sensation in the distal half of the fore limb, up to a distance of 6 or 7 inches above the carpus. This operative anaesthesia is of value in diagnosis and treatment of lameness and allows neurotomies, tenatomies and other operations below these injections. The following will illustrate.

Bay Mare. Right anterior limb, injected one and one-half grains of cocaine in 5 c.c. sterile water over the ulnar and median nerves.

Bay Gelding. Right anterior limb, injected 10 c.c. of a 1 per cent. solution of novocain over the ulnar and median nerves.

Anaesthesia was complete in each case at the end of twelve minutes, and gave an operative anaesthesia from 6 inches above the carpus to the hoof.

External and Internal Metacarpal or Plantar Nerves. These nerves should be blocked at the same time, there being no advantage in narcotizing but the one side. The site of injection lies from immediately above the fetlock to the middle of the metacarpus, in the groove on each side of the flexor tendons. Complete enervation of the region below the injection is obtained, allowing such operations to be performed as high and low neurotomies, firing, and all surgical procedures on the foot. The following examples will illustrate:

Gray Mare. Right anterior limb, injected one grain of cocaine in 5 c.c. of sterile water over each nerve.

Bay Gelding. Left anterior limb, injected one grain of cocaine in 5 c.c. of sterile water over each nerve.

Bay Gelding. Right anterior limb, injected one grain of cocaine in 5 c.c. sterile water over each nerve.

Black Mare. Right anterior limb, injected one grain of cocaine in 5 c.c. sterile water over each nerve.

Results in all cases were apparent after seven minutes, and gave a complete anaesthesia below the point of injection.

Digital Nerves. As in the case of the above nerves these injections should be made on both sides at the same time for complete anaesthesia. The site of injection is between the fetlock joint and the coronet, in the furrow between the posterior border of the first phalanx and the anterior border of the flexor tendons. This gives a good anaesthesia of the foot, and can therefore be utilized for operations upon its structures, and for digital neurotomy.

Gray Mare. Left anterior limb. Injected one grain of cocaine in 5 c.c. sterile water over each digital nerve.

Bay Gelding. Left anterior limb. Injected 8 c.c. of 5 per cent. novocain over each digital nerve.

Black Gelding. Right anterior limb. Injected 10 c.c. of 1 per cent. solution novocain over each digital nerve.

Bay Gelding. Left anterior limb. Injected one grain of cocaine in 5 c.c. of sterile water over each digital nerve.

Results were apparent at the end of twelve minutes in all cases, and a good anaesthesia was present below the point of injection.

2. *Pelvic Limb.*

Posterior Tibial Nerve. The most advantageous point for injection is on the median side of the limb, a short distance above the hock, in the space between the deep flexor and the anterior border of the tendon Achilles. The results obtained from the injection of this nerve are fairly good; operative anaesthesia is present at the point of injection, and a marked loss of sensation is noticed on the inner side of the hock, which may be valuable in the treatment of spavin lameness, diseases of the tendons, etc.

Bay Mare. Left pelvic limb. Injected two grains of cocaine dissolved in 10 c.c. sterile water over this nerve.

Bay Mare. Right pelvic limb. Injected two grains of cocaine in 10 c.c. sterile water over nerve.

Gray Mare. Left pelvic limb. Injected 10 c.c. of 1 per cent. solution of novocain over nerve.

Bay Gelding. Right pelvic limb. Injected 10 c.c. of 1.5 per cent. novocain over nerve.

Bay Gelding. Right pelvic limb. Injected 10 c.c. of a 1 per cent. novocain solution over nerve.

Results were noticeable in twelve minutes in all cases, and a marked loss of sensation was noted on the inner and posterior part of the hock.

Anterior Tibial Nerve. This nerve is reached by deep injections between the lateral and anterior digital extensors, at a point 8–10 cm. above the flexure of the hock. Infiltration above the nerve gives a decrease in sensation on the anterior face of the tarsus, and on the anterior and outer faces of the metatarsus. The following experiments are typical:

Bay Gelding. Left pelvic limb. Injected two grains of cocaine in 10 c.c. of sterile water over the anterior tibial nerve.

Bay Mare. Left pelvic limb, injected two grains of novocain in 10 c.c. of sterile water over the anterior tibial nerve.

Bay Gelding. Right pelvic limb, injected 10 c.c. of 1 per cent. novocain over the anterior tibial nerve.

Grey Gelding. Right pelvic limb, injected two grains of cocaine, dissolved in 8 c.c. of sterile salt solution, over the anterior tibial nerve.

Marked loss of feeling in the above mentioned areas was present in all cases within twelve minutes.

Anterior and Posterior Tibial Nerves. For practical application these nerves are best injected together, thereby gaining an operative anaesthesia of the whole limb below the point of injection. This allows all operations for spavin, lameness, tenatomies, firing for ring bone, and all surgical procedures upon the hock and digital structures. The following will illustrate:

Gelding. Right pelvic limb, injected 10 c.c. of 1 per cent. solution of novocain over each of the above nerves.

Gray Mare. Right pelvic limb, injected one and one-half grains of cocaine dissolved in 10 c.c. sterile water over each of the above nerves.

Anaesthesia in each case was complete at the end of eight minutes and extended from the point of injection to the foot.

Plantar and Digital Nerves. From repeated observations it is evident that the injection of these nerves, and the results to be obtained, are identical with those of the thoracic limb and no further remarks are considered necessary regarding them.

General Conclusions

The writer would present the following general conclusions:

1. That local anaesthesia is an important type of anaesthesia in veterinary surgery which should be more often brought into use by the practitioner.

2. That its use alone, or in conjunction with narcosis, should be substituted for general anaesthesia whenever possible.

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A SURVEY OF THE INTESTINAL PARASITES OF THE DOG

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The object of this investigation was to gain some definite knowledge of the kind, intensity and frequency of intestinal parasites of the dog with particular reference to the Nematodes and so far as possible to check the degree of infestation with the clinical manifestation. No discrimination was made. Each dog and the feces of each dog which entered the clinic after December 8, 1916, was examined. Therefore these results quite accurately indicate 1, the percentage of dogs infested in the vicinity of Ithaca; 2, the variety of parasite; and 3, the relative degree of infestation.

In examining the feces the following technic was followed: the feces were mixed with physiological salt solution, strained through wire gauze to remove coarse particles, and washed in a test tube. The turbid liquid was then centrifuged from three to five minutes. Some of the sediment was then drawn up with a pipette, placed upon a glass slide and examined under the low power of the microscope.

The parasites found in this vicinity were:

(1) Anklystomum trigonocephalum, (2) Ascaris marginata, (3) Tricocephalus depressiusculus.

The following table gives the results of the examination.

Number of cases examined.....	150		
Number of dogs infested.....	98	per cent.	65.33
Number infested with Anklystomum...	46	per cent.	30.66
Number infested with Ascaris.....	35	per cent.	23.33
Number infested with Tricocephalus...	17	per cent.	11.33

In addition to the above table it should be mentioned that the eggs of the whip worm (Tricocephalus depressinsculus) were never found alone but always in conjunction with those of the hook worm (Anklystomum trignocephalum). Consequently it has been impossible to arrive at any conclusion with respect to the

symptoms caused by these parasites. The table also does not include the number of animals which were infested by more than one variety of parasite.

It was also found that the relative number of eggs found in the feces gave absolutely no indication of the condition of the host. Many of the most emaciated cases of hook worm disease showed relatively few ova in the feces, while many of our most thrifty and vigorous patients were apparently the more heavily infested.

GARBAGE FEEDING, AND THE CARE OF GARBAGE FED SWINE

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The present scarcity of meat and meat products is being forced to our attention by constantly rising prices. At the same time the grains that formerly have been utilized to produce meat have risen so rapidly in price, and are so urgently needed as human food, that the effect has been to curtail the fat stock industry at a time when there is an acute demand for the products of the fattening pen. The situation requires that substitutes for grain shall be utilized to the utmost in producing meat, and the purpose of this paper is to point out the value of city garbage in pork production, and to show how it can now be utilized to a much greater extent than has been possible in the past. Incidentally it is hoped that some light may be thrown on the question of garbage disposal from the standpoint of city economy, a problem that is claiming close attention at the present time.

In the past, the practice of feeding city garbage to hogs has met with varying degrees of financial success, and consequently one finds widely divergent views regarding the feasibility of using it profitably in this manner. Although it is true that some men have been able to make the practice pay exceedingly large profits, we are on the other hand reminded of the fact that others have failed to reap any profits whatever. We may almost conclude that failures have been the rule, anyhow the fact remains that in our State only a small proportion of city garbage is fed to swine.

Hog cholera has been the cause of many failures, for it has occurred so frequently and introduced such an element of pure luck, that feeders have been exceedingly cautious in regard to assuming risks incident to feeding large numbers of hogs on city garbage. As a result, undertakings of this kind have been limited in scope, and most of them have been conducted by haphazard hand-to-mouth methods that did not permit large gains even when hog cholera did not appear.

Because it has now been well demonstrated that serum can be relied on to prevent almost all losses from cholera, and because pork has more than doubled in price since the war began, new financial opportunities, and opportunities for a most effective form of food conservation have been brought about.

During the past few years we have vaccinated many hogs for men who feed city garbage, and a considerable number of these herds have yielded rich financial returns. On the other hand, the causes of the failures observed have been so obvious, and so easy to overcome, that an effort has been made to gain more insight into the problem as a whole, and to seek out the reasons why more city garbage is not utilized as feed.

In following out this idea a questionnaire was sent, in November, 1915, to the mayors of fifty cities in the State, and the answers received give interesting and valuable information on the point under consideration.

Question No. 1. Is your city garbage an asset or a liability? There were thirty-eight definite answers: twenty-eight, liability; one, asset; nine, garbage removed by contract of private individual with property owners. Under these circumstances it is in reality a liability, for the city residents pay to have the garbage removed. Most of the mayors that mentioned these contracts with property owners expressed the view that they were unsatisfactory.

Question No. 2. Is your garbage removed by contract? (Meaning contract of private individual with the city.) Twenty-seven mayors answered in the affirmative, eight in the negative. This means that most of our cities do not assume final responsibility for garbage collection and disposal. The work is done according to municipal regulations, but not by men in the employ of the cities.

Question No. 3. Are the contracts let each year or for a term of years? There were twenty-four definite answers divided thus: Yearly contracts, ten; two-year contracts, four; three-year contracts, six; five-year contracts, four. Thus it is shown that short-term contracts predominate, and it is obvious that they are unsuited to the practice of feeding the garbage to hogs. A feeder can scarcely afford to make adequate preparations for handling his hogs unless he can be assured that his supply of feed will not be cut off shortly after these preparations are made. It seems

very likely that short-term contracts may be responsible in no small measure for the fact that more garbage is not fed, and also for the fact that in some instances where it is fed, preparations are so meager as to eliminate profits or render the practice a public nuisance.

Question No. 4. Are ashes and rubbish included in your garbage contract? There were twenty-nine answers: nineteen negative, eight affirmative, one including ashes only, and one including rubbish only. These answers indicate that as a rule ashes and rubbish are not included in the garbage contracts. This is as it should be, for it is undoubtedly true that there are some who will now take garbage contracts who would hesitate to do so were they required also to look after collection of ashes and rubbish.

Question No. 5. What disposition is made of your city garbage? There were twenty-nine answers to this question, divided thus: Dumped, seven; fertilizer, seven; fed, seven; reduced, four; burned, two. In approximately one-fourth of the cities that turned in answers the garbage was fed, and as the answers to question No. 1 indicate, those who feed it are paid to collect and remove it from the respective cities. This does not mean, though, that one-fourth of the garbage in the State is fed, for the cities that reported their garbage as being fed were all comparatively small.

One mayor who reported that the garbage from his city was being used as fertilizer enclosed a letter from which the following paragraphs are quoted:

"I have answered the questions you have asked and can think of no other additional facts which would interest you. If you can devise some manner to feed garbage to swine without danger we would certainly be glad to hear from you, as we are considering making collection and disposal of garbage a municipal function.

"The people who have had the contract for removing our garbage have tried this out, but with considerable loss, as the hogs contract cholera and disappear like a June frost.

"Let us hear from you when you have completed your work."

Question No. 6. What does it cost annually to remove the garbage from your city? There were twenty-six answers to this question showing these figures: three cities paid less than $1,000 annually; ten cities, $1,000 to $5,000; eight cities, $5,000 to

$20,000; two cities, $20,000 to $100,000; and three cities paid more than $100,000 annually for collection and disposal of garbage.

These figures, together with other information which has been secured, indicate that it is the custom for cities to let their contracts so that the sum allowed the contractor will cover all costs of collecting and hauling. Then if the contractor can utilize the garbage as feed for hogs, a very great proportion of the selling price of the pork produced will be profit.

Question No. 7. Is a considerable amount of garbage removed by persons other than the one to whom the contract is let? There were twenty-four answers to this question: fourteen in the negative, and ten in the affirmative. These answers indicate that some property owners dispose of their garbage by private arrangement even when there is a city contract. Hotel and restaurant owners make up a considerable proportion of this class, and it is undoubtedly true that most of them are paid for the garbage.

Question No. 8. How often do you require the garbage to be removed from each street? There were thirty replies to this question. Six cities required collections once weekly; eight, twice weekly; four, three times weekly; two, daily; five, once to twice weekly; three, daily to twice weekly; two, daily to once weekly. These answers show that, in general, the garbage is collected while it is sufficiently fresh to be utilized to advantage in feeding swine.

Further information obtained principally from a recent report of the Director of the New York State Bureau of Municipal Information, indicates that feeding is the only truly economical method of garbage disposal in cities of less than 100,000 inhabitants. It is an open question whether it is best to maintain municipal piggeries or to have the garbage removed by contractors who maintain their own establishments. Municipal piggeries have not, as a rule, been wholly successful, and some have been flat failures, but there are exceptions that prove the rule. Hog cholera alone has rendered such enterprises very uncertain, but there is the additional fact that they require exceedingly careful supervision, and it is difficult to secure this by employing untrained men who have no direct financial interest in the success of the enterprise.

According to reports, Worcester, Massachusetts, is maintaining a very successful piggery. In 1914, a special commission

appointed to investigate conditions there reported: "That the disposal by feeding is the most economical method, the greatest intrinsic value of the garbage, the feeding value, is made use of; that the garbage of Worcester cannot only be disposed of without cost but the revenues from the sale of hogs have been almost sufficient to pay for collection." Worcester is a city of about 150,000 inhabitants and judging by costs of collection and disposal in other cities of the same size, it must be saving itself at least $40,000 annually by maintaining a municipal piggery. Better still, it is helping to solve the nation's food problem.

This special commission goes on to assert that out of 2,276 hogs sold in one year only eleven failed to pass federal inspection, and it makes the further observation that the wide difference of opinion regarding the possibility of feeding garbage profitably is due to the varying degrees of efficiency with which sanitary conditions at the piggeries are maintained, a statement which our own experience leads us to believe is decidedly to the point.

We have already mentioned a few factors that determine success or failure in regard to feeding city garbage as a private enterprise, but in this connection, in order to show something of the profits obtained under some circumstances, it is only necessary to state that the New York Medical Journal, 1912, is authority for the statement that the garbage collected at Grand Rapids, Michigan, at a cost of $26,000 annually is sold to a contractor at forty-five cents per ton. This contractor sold more than 10,000 hogs annually for which he received $135,000, and also secured fertilizer valued at $36,000. Our own estimate — counting that 7,500 was the average number of hogs on hand at one time, and that a ton of garbage furnished daily feed for seventy-five — is that this man paid only about $16,500 annually for feed. Whether he raised the pigs or purchased them as shoats he made a handsome profit. In our personal experience we know of several men who have reaped corresponding profits on a smaller scale during a term of years, and of others who could easily have done so had they not failed to realize the importance of keeping their herds immune. These are simply examples of what is being done in some instances, and a suggestion of unaccepted opportunities that exist in other places.

According to a recent report of the Canadian Veterinary Direc-

tor General the value of garbage as a feed for hogs is recognized by the Canadian government. In order to control the sanitary conditions at the piggeries, the government requires those engaged in feeding garbage to secure licenses, and it plans to issue these only to men who have necessary equipment, and who maintain their piggeries in such manner as to prevent them from becoming public nuisances.

There is much more that might be said on the subject, but in this paper the essential points to which it is desired to direct attention are that garbage can be fed to immune hogs with reasonable safety, that the practice can be made to yield large profits, and that so far it has not claimed the attention it deserves. It is well to add, though, that no city should undertake the establishment of a municipal piggery unless the enterprise can be assured of constant and skillful lay and veterinary supervision. On the other hand if a city plans to dispose of its garbage by contract, municipal regulations requiring that the garbage shall be properly drained, that no rubbish shall be included in it, that the contractor shall have exclusive rights to collect, and that the garbage shall be made available to the collectors at regular stated intervals, will add greatly to the advantage with which a city can let its contract. Finally, a long-term contract, or a short one with optional renewal on the part of the contractor, will interest many who are not now willing to accept the uncertainties incident to one or two-year agreements.

The general preparations necessary for maintaining a herd on garbage need not be expensive, and under careful management, quick returns can always be expected. Whether the herd to be kept is large or small it is a great mistake to keep more animals than can be well fed and housed. In selecting a locality for a piggery, well drained soil, plenty of room, water supply, natural shelter in the form of woods or hills, proximity to the city from which the supply of garbage is to be secured, and the character of the road leading to the city are all prime considerations.

Where large numbers of hogs are to be fed during winter, tracks and cars for distribution of feed, concrete feeding platforms, clean and dry sleeping quarters, room for exercise, facilities for disposing of unconsumed material, caldrons for heating frozen or

stale garbage, and water pressure for flushing out the pens are all desirable. Nevertheless much less expensive equipment can be made to serve until the enterprise itself will pay for needed improvements. In sheltered localities a number of roomy yards, each containing a portable hog house and a wooden or concrete platform on which to feed, can be made to serve admirably.

Methods of swine husbandry. No attempt will be made in this paper to outline a complete system of swine husbandry, but there are certain essential points quite often wholly neglected, any one of which may easily mean the difference between success and failure, and of these, brief mention seems desirable.

The question whether to raise or purchase pigs for feeding is one that local conditions must decide. As long as the country near the piggery will furnish the needed number of young shoats at a reasonable price, it is very doubtful if a beginner should attempt to breed pigs when garbage only is to be fed. In normal times it is much simpler to purchase and care for young shoats, and more profitable in the long run, in all cases where the very best quarters cannot be provided, and where experienced and reliable help is not available at all times. Under some circumstances it is of advantage to secure small feeding shoats that have been shipped to stockyards, but there are grave dangers connected with this practice. These will be discussed in the portion of this paper dealing with veterinary supervision of the piggeries.

Just now, when the high price of pork is driving large numbers of brood sows to market at a time when they can ill be spared, it is certain that whenever suitable quarters and proper care can be provided, raising the pigs instead of buying them, will in most cases be the most certain method of insuring a plentiful supply of feeders.

Although we have seen fine pure bred hogs raised on garbage alone it is doubtful if this is to be recommended in most cases. But when an even, well graded herd can be maintained at the price of purchasing a good pure bred sire every year or two, such an investment is well worth while. Size, hardiness, prolificacy, and a moderate degree of refinement, rather than show ring niceties, are the characteristics to be sought. The breeding herd should consist for the most part of old sows of well established

breeding merit. These breed with greater certainty, farrow larger litters, and nurse their pigs better than young sows.

There are several points in regard to feeding that are commonly overlooked. It is always to be remembered that the critical point in a pig's life is from weaning time until a weight approximating fifty pounds is reached. It is during this period that overfeeding is most likely to take place, and this is especially true where garbage is fed, because pigs have to develop an appetite for it, and because the supply is often so plentiful that an effort is made to make the pigs eat as much as possible. Carefully checked experiments have shown repeatedly that a pig once overfed while young subsequently requires much more feed per pound of gain, nor can he be made to gain nearly so fast under any circumstances as the pig that has had his diet more carefully regulated.

Prior to weaning, a pig is accustomed to taking his feed in small quantities several times daily. A sudden change from this routine to that of one or two feeds a day, and a radical change in the character of the feed at the same time, will surely lead to trouble, especially if garbage is the sole diet, and if it is not given with discretion.

Supplementary feeds in the form of pasture, soiling crops, a little grain, and under some circumstances, skim-milk or whey, will much more than pay for themselves if given in limited quantities to pigs during the first few weeks following weaning. Pigs must be kept growing and vigorous while young if they are subsequently to utilize their feed with a degree of efficiency approaching the maximum. Cooking the garbage that is fed to young pigs is often a good practice, and is to be recommended where supplementary feeds are not furnished.

Pasture in summer is a great asset, because it supplies cheap nutriment, acts as a tonic, and induces the pigs to take exercise. In some places it is the custom, in summer, to feed on the ground in an open field, and plow the unconsumed material under at frequent intervals. The advantages of this practice are obvious.

Considerable numbers of hogs may be kept in the same enclosure during warm weather but it is usually best to grade them

according to size. In winter time special precautions are necessary. Too much emphasis cannot be placed on the importance of dry sleeping quarters, and on provisions to prevent pigs from piling up at night. When large numbers of pigs are kept together in cold, damp quarters, they frequently form the habit of piling up to keep warm, and an occasional pig is smothered, but this represents only a small portion of the loss. The worse loss comes from the alternate heating and cooling and the attending ills. A pig becomes cold and forces his way under the others in order to get warm. Then when he is half smothered and superheated he fights his way out and his steaming body is suddenly exposed to the cold air. This process is repeated several times daily, with resulting coughs, pneumonia, and general unthriftiness. Shoats that have attained a weight of seventy-five pounds or more will endure a surprising degree of cold, if they are kept dry.

A final consideration which is frequently neglected is the desirable age at which to market hogs. The common custom is to hold them far too long. When the average well fed hog is about nine months of age he ceases, quite suddenly, to make large or economical gains. Very few men appreciate this fact, and consequently most feeding hogs are kept weeks and months after they should be sold and replaced. Perhaps the best single standard by which to measure profitable swine husbandry, is the age at which the animals are finished and ready for market.

Veterinary supervision. The fact that garbage fed hogs are in great danger of hog cholera infection due to virus contained in pork trimmings has been demonstrated by careful experiments, and clinical observations bear out the evidence. This danger has proved to be so great that we believe every garbage feeder who consults his veterinarian should be advised to begin at the earliest possible time to maintain an immune herd. These men should be given correct information concerning the danger involved in immunizing, and the probable cost. They should also be told that when once they begin they must continue if they expect to maintain their herds in safety.

The system to be followed in maintaining an immune herd where the practice is to purchase all pigs is comparatively simple.

These animals should receive serum alone before they are transferred if they are to pass through public stock yards or to be placed in cars; otherwise the serum may be given after the transfer is made. Four or five weeks after the serum is given, simultaneous treatment is administered and a permanent immunity is established.

Any man contemplating the purchase of pigs that have been shipped to stock yards should be advised to watch the cars as they are unloaded, and buy only from cars that contain no animals dead or visibly sick. The pigs should be purchased as soon as possible after being unloaded; then they should be given serum alone without delay and shipped to their destination.

Numerous disasters have been reported as a result of purchasing pigs that have been in the yards several days, and either neglecting to give serum at all or giving it too late. It must be remembered that young pigs are often sent to the yards because they have already been exposed to hog cholera at home. Then a trip by rail with its unavoidable hardships, and several days in the yards with daily exposure, often places these pigs in such condition that nothing will prevent complete disaster. They are in the early stages of hog cholera before they receive serum.

In places where brood sows are kept and pigs are raised the veterinarian's task is to establish and maintain immune herds. The establishment of an immune herd is often attended with considerable difficulty. The first cost is heavy because there are often so many large animals, the breeder is often a little skeptical, and it is sometimes difficult to tell him of the dangers incident to simultaneous treatment without causing him to magnify these dangers far beyond their real proportions. Nevertheless, the danger should always be mentioned before treatment is administered, and our clients should be informed that during the month following simultaneous treatment, none of the treated animals are to be sold, and neither are new purchases to be added to the herd without receiving treatment.

The herd to be immunized often presents peculiar difficulties, because it so frequently consists of a miscellaneous collection of hogs of all sizes and ages, and in all conditions. The general

plan followed is to give simultaneous treatment to all animals that are in condition to receive it, and at the same time to give serum alone to those which on account of small size, poor condition, late pregnancy, or recent farrowing are not fit subjects to receive virus. These are rendered passively immune until the time arrives when they may receive simultaneous treatment in comparative safety.

When a herd immunity is once established the problem becomes greatly simplified. The pigs of immune sows are less likely to contract cholera at an early age, and they can be immunized while comparatively young, at a time when they are easily handled, when the cost of serum is light, and when the risks incident to immunization are at a minimum.

Considerable experimental work must yet be done before we can say with certainty just what is the best system to follow in immunizing young growing pigs, in herds where hog cholera virus must be regarded as being constantly present. We must content ourselves, at present, with outlining a system that has given excellent results throughout a term of years.

Briefly, this system consists of castrating the pigs when they are about four weeks old, giving serum alone at four to six weeks of age, weaning at seven to eight weeks of age, and giving serum and virus at nine to twelve weeks of age. In following this system it is presupposed that active infection is not present — that is, that there are no sick pigs in the herd — and that the animals are maintained in good growing condition. For purposes of identification we have found it convenient to notch the left ear when serum alone is administered, and the right when the animal receives simultaneous treatment.

This general system has to be varied according to circumstances. Sometimes pigs of immune sows will not live four weeks before they begin to contract cholera and in these instances serum alone must be given as soon as the disease appears. Cases have also been reported to us in which herds of pigs did not remain immune a month after receiving serum, but we have observed this only in a few individuals — never in a considerable portion of a herd. Here again, the appearance of cholera would call for immediate serum treatment.

As a rule, it will be found advantageous to delay giving serum alone to the young pigs as long as it can be delayed in comparative safety, and also to delay simultaneous treatment until the pigs have attained a weight of fifty pounds, but in either case careful watching is essential. In whatever way the system is varied, pigs should not receive virus at castrating or weaning time, or while they are generally unthrifty. Neither should they receive it while they are so young that a permanent immunity will not be established.

The technique of serum treatment appears so simple that essential points are sometimes overlooked. In treating young pigs special care is required. The dose should be divided, the inner region of each ham receiving half the amount required. Then the ham should be kneaded gently a moment to prevent encapsulation and aid absorption. Dry quarters should be provided for the pigs during the first few days following treatment. Except in severe weather it is well to work out of doors, and in all cases the serum receptacle should be covered to prevent dust contamination. In injecting animals too large to be thrown conveniently, the needle should be inserted quite deeply and in a downward direction into the loose tissue immediately behind the ear. Unless the serum flows freely from the syringe the position of the needle should be changed until but little force is required in making the injection. Here again, vigorous massage following the injection will help to prevent encapsulation which is especially likely to occur when it is found necessary to inject behind the ear. Injecting high in the neck is to be avoided for the dose is likely to find lodgment in the thick layer of fat immediately beneath the skin at this point, and rapid absorption cannot be expected.

Careful attention to these seemingly trivial details, together with special precautions regarding the surgical cleanliness of instruments and site of injection, will eliminate much danger from abscesses, and insure more prompt and effective action on the part of the serum.

In conclusion, let it be said that we are fully aware that a large portion of this paper has been given over to discussion of matters which some may not consider as belonging strictly to the veterinary profession. Nevertheless it has seemed worth while to direct

attention to the obligations resting on some of our cities to work out systems by which their garbage may be utilized as feed, and to show something of the financial opportunities that await the men who undertake to maintain hogs on city garbage. These enterprises represent a most effective type of food conservation and are especially important in States having large urban populations; they can prosper only under skillful veterinary supervision, and a comprehensive understanding of the situation as a whole is essential to all members of our profession who are called to render this service.

OROKINASE AND PTYALIN IN THE SALIVA OF THE HORSE

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The literature upon this subject has been rather thoroughly and painstakingly reviewed in three papers of recent publication. Palmer, Anderson, Peterson and Malcomson (1), R. J. Seymour (2) and Hayden (3) have reported upon different studies concerning salivary digestion in the horse. It is the purpose of this paper to submit data gathered more recently upon the subject and to compare the results with those reported in the preceding work. Palmer (1) and his co-workers report the existence of an enzyme, present in the buccal and possibly the lingual glands, that activates a ptyalinogen present in the secretion of the salivary glands of the horse. They conclude from the evidence of their data that neither saliva obtained from a parotid fistula nor a glycerine extract of the parotid gland digest starch. They also show that the mixed mouth secretions obtained from an esophageal fistula are more powerful in their amylolytic action than those obtained from the mouth. The name orokinase is given to the activating enzyme which activates the parotid saliva and the claim is made by them that the enzyme is present in the mouth secretions of both horse and man.

In Seymour's work mixed saliva of the horse is shown to have produced reducing sugar after eight hours' digestion. The parotid saliva does the same thing with slightly accelerated action. Old and recent glycerine extracts of the salivary glands of the horse are negative up to eighteen hours. Water extracts were positive after six hours' digestion with a 2 per cent. corn starch solution. There is no evidence of an enzyme in the mouth secretions that activates a ptyalinogen present in the secretions of the salivary glands. The saliva of the horse, both the mixed and the isolated secretions of the parotid and submaxillary glands, contains a diastase capable of converting starch into sugar. The diastase is extremely feeble, requiring at least five hours' digestion for the conversion of starch to reducing sugar.

Hayden (3) after having digested a 1 per cent. solution of soluble starch with parotid fistula saliva up to twenty-four hours obtained evidence of the presence of an enzyme capable of carrying the digestion of the 1 per cent. starch solution to the maltose stage at least. The test for sugar was made with Benedict's quantitative solution.

Carlson, Greer and Becht (4) collected mixed saliva from two horses using pilocarpine as a sialogogue. They report no amylolytic action on the part of those samples. Twelve other samples of mixed horse saliva collected by them showed no power to digest starch.

Carlson and Crittendon (5) inserted a canula into Stenson's duct. The parotid saliva collected by that method digested starch. The saliva was collected from a human subject.

M. Foster (6) in discussing the action of the saliva of man says that the parotid saliva alone digests starch and that the secretion of the submaxillary gland is sometimes more powerful than that of the parotid.

Prof. E. H. Starling (7) reports the stimulation of the flow of saliva in the dog by the use of pilocarpine with no amylolytic action on the part of the saliva.

Wiley (8) in giving an analysis of the grains says that there is in corn 71.48 per cent. of starch, sugar and dextrin combined. Maize flour contains 78.36 per cent. of starch and sugar. Unhulled oats contain of starch and sugar 57.93 per cent. Hulled oats contain 67.09 per cent. of these substances.

Another author (9) claims that there is practically no dextrin or maltose in untreated grains.

Bradley and Kellersberger (10) report the presence of an active diastase in the seed of both mature and young corn. Jordan (11) says that sugars are formed in small quantities in the hays and in scarcely appreciable quantities in the grains. Saccharose exists in considerable proportion in field corn. Maltose exists in no quantities. Dextrose is found in maize.

Effront-Prescott (12) state that corn, rye and all other cereals contain considerable quantities of amylase and other substances that accelerate diastatic action. The action of the diastase begins during the process of milling. There is a transformation of the

starch of the grains into sugar and the action of the amylase is shown at grinding. The amylolytic action takes place more readily in the presence of water.

Methods

With a desire to corroborate as far as possible the conclusions reached by Palmer (1) and his co-workers we have worked along lines very similar to those followed by them. Palmer (13) says that pilocarpine will digest starch and that enough of it will be eliminated in the saliva to show that effect. He claims that such being the case pilocarpine cannot be used to stimulate the flow of saliva for the purposes of this investigation. Hayden (3) in previous work used pilocarpine as a stimulant to secretion. It was used in two forms, pilocarpine hydrochloride crystals and Strasenburgh's hypodermic tablets. No record was kept in that work of the kind used for the stimulation of any particular secretion. We thought it best to confirm Palmer's (13) results and so have tested a few samples of pilocarpine in both forms. For all the digestive work done in our experiments a 1 per cent. solution of corn starch was used. Five c. c. of the corn starch solution was digested with whatever substance was under observation. With most samples digestion was carried on for two hours in an electric incubator at a temperature of 38–40 degrees C. A sample of the starch solution was carried along with every set of samples digested and then tested for reducing sugar. If reducing sugar appeared in the starch the quantity was subtracted from every sample showing sugar in that particular series. An accurate report of the sugar actually produced by the action of any of the substances tested is thus given in the tables. Sugar appeared in the starch solution but twice in all the different series we carried through. The starch solution was made up fresh every two or three days and so results were in no way compromised by the use of an old solution. In the pilocarpine series we tested pilocarpine alone, pilocarpine plus starch and starch. Any reduction occurring from the starch alone or the pilocarpine alone could be recognized and subtracted from the results obtained from pilocarpine plus starch. In this manner we hold that the actual amount of digestion brought about by the action of the salt can be stated with fairness and accuracy. The same thing holds true for the other substances tested for the presence of an enzyme digesting starch.

The test for reducing sugar was made with Benedict's single solution for quantitative work. The solution was standardized by a 1 per cent. solution of pure dextrose (Merck's). In the determination of sugar 10 c. c. of the reagent was measured into a beaker from a burette. The reagent was brought to the boiling point and then the digested material, or a portion of it, was added. If the reduction was not complete the 1 per cent. dextrose solution was added a few drops at a time from another burette and the mixture boiled for one-half minute between each addition. Benedict's solution gives a white color when all the copper has been reduced and thus the end point is very easily determined. We used 10 c. c. of the reagent because of the scarcity of some of the ingredients.

The parotid saliva was collected from a fistula. It was tested for reducing power within itself. No sample of parotid saliva showed reduction that could be measured by the reagent used. Parotid saliva was digested with 5 c. c. of starch, with starch in the presence of two drops of mixed horse saliva in a dilution of 1–10 and with mixed human saliva in the dilution 1–50 and the same quantity. The purpose of these tests was to verify the presence of orokinase in mixed horse saliva and in mixed human saliva. The samples in these two series were digested for two hours. Some samples of parotid saliva were digested with starch up to twenty-four hours. In the tubes digested for twenty-four hours 1–50 c. c. of saliva was used. The samples of saliva were all collected fresh in the morning of the day in which the series of tests were made. The parotid saliva was collected while the animal was manger or trough feeding. The flow of mixed horse saliva was stimulated in different ways. Some methods of stimulation were mechanical. Some samples were stimulated by hypodermic injection of arecoline. Arecoline was also tested as was pilocarpine to show its reducing power and action upon starch. It was found negative in both respects. Some of the samples of mixed saliva flowed freely, some did not. Our data thus undergo the same limitations as do those of Palmer et al (1). Mixed saliva was tested for reducing power. Its digestive action was sought upon cooked starch, upon the raw starch of corn and oats and upon cooked starch in the presence of parotid saliva and mixed human saliva.

Corn and oats were ground up in a food chopper and then mixed with water. The filtrate passed through cheese cloth was tested for reducing power using Benedict's quantitative reagent. These separate grains were digested with parotid and mixed saliva of the horse and mixed human saliva. At the end of the digestion period they were filtered and a portion of the filtrate tested for reducing power. The division of the filtrate into portions was necessary because some samples contained so much reducing sugar that they could not be measured using 10 c. c. of the reagent. This condition occurred in samples that were digested alone as well as in those that were digested with saliva. The amount of reduction is indicated in the tables on the basis of the whole filtrate.

Mixed human saliva was collected from various individuals. Chewing of parowax was resorted to to stimulate the flow of the samples. The action of mixed human saliva in the dilution of 1–50 was tested upon starch alone, upon starch plus mixed horse saliva and upon starch plus parotid fistula saliva. The activity of the saliva was also tested upon the raw starch of corn and oats and upon cooked starch.

For all samples the tests used to indicate the extent of digestion were the amount of clearing of the starch solution, reaction of the starch to N/20 iodine and the quantitative tests for sugar. Clearing of the starch solution in this work has not proven to be a very satisfactory index. Most of the samples have shown a considerable opalesence at the end of digestion and we have indicated the appearance by the letter o. Different reactions to N/20 iodine have been indicated as follows: erythro-dextrin by R, achroo-dextrin by —, G. for a good reaction, S for a slight reaction and so on. These changes will indicate some digestive action. A very active enzyme should produce an amount of sugar that would give an appreciable amount of reduction.

Extracts of the parotid, submaxillary, sublingual, buccal, lingual glands and of the mucosa of the buccal region were made with a 50 per cent. glycerine solution. These extracts were made from four different horses. The extracts were made on the day during which the animal was killed and digestion was carried out the next day. Material was obtained from one esophageal fistula.

More data need to be obtained from these last two sources to make results conclusive.

Results

Pilocarpine and arecoline both act as sialogogues. In an effort to verify the statement that pilocarpine digests starch we have tested some samples of pilocarpine crystals and hypodermic tablets. Five-tenths grain of each was used. The solution in each case was made up to 10 c. c. and 2 c. c. of the quantity tested. Arecoline was in the form of hypodermic tablets 0.5 grain and was tested in the same way. Results are indicated in table 1. The hypodermic tablets of both pilocarpine and arecoline contain a reducing sugar. This fact is established by testing the water solution alone for reducing power. Neither of the substances showed starch digesting power. Solution 0.5 grain of pilocarpine hydrochloride crystals show no reducing power either when tested alone or after having been digested for two hours with the 1 per cent. solution of corn starch.

Tables 2, 3 and 4 present data which should show the presence of orokinase in the human saliva. Table 2 shows the digestive action on 2 drops of human saliva in a dilution of 1–50. Digestion was carried on for 2 hours. In no case was the solution clear in that time. N/20 iodine gave reactions in which the color was good as indicated by G to the erythrodextrin stage as indicated by R. Those samples with reaction G are in the minority.

The erythrodextrin stage is very frequent in the series. Reducing sugar was found in quantities up to 0.032 grams. Most of these reactions would give a ready reduction with Fehling's solution. One sample in a dilution of 1–100 gave 0.005 gram of sugar. The record of the influence of human saliva in a dilution of 1–50 upon mixed or parotid saliva of the horse does not indicate any activation. The average amount of sugar is no greater than that obtained from the action of the human saliva alone. Neither is the evidence as shown by the clearing of the starch solution or by the reaction to N/20 iodine in support of the presence of an activating agent in the human saliva. Table 4 gives no evidence of activation. The starch solution is not appreciably cleared in any case. Reaction to N/20 iodine remains the same as indicated in tables 2 and 3 and the amount of sugar measured is

no greater. The evidence as shown in these tables in favor of a kinase is negative.

That mixed horse saliva digests cooked starch as readily as mixed human saliva is not borne out by the data submitted in tables 5 and 6. In neither series did the starch solution completely clear in two hours. Human saliva carries the starch to the achroo-dextrin stage in that time. Mixed horse saliva rarely carries the digestion beyond the regular starch reaction to iodine. The amount of sugar produced as a result of the action of human saliva is large. The mixed horse saliva produced negligible quantities of sugar except in three cases and they are in no way comparable with the average three cases in the mixed human saliva.

Mixed horse saliva from the mouth in a dilution of 1–10 and two drops used has not shown any activating power when digested with parotid fistula saliva from the horse. Tables 7 and 8 compare the action of the diluted horse saliva and its admixture with the parotid saliva. Neither indicates a clearing of the starch solution. The reaction to N/20 iodine is G in both cases with one exception in each. It is R once in table 7 and F once in table 8. The maximum amount of sugar produced is 0.007 gram in each case. If the action indicated in these tables is typical the conclusion cannot be reached that there is a kinase in the mixed saliva of the mouth of the horse that activates the parotid saliva from that animal.

Palmer et al (1) state that the raw grains of corn and oats contain no reducing sugar that will show when a test is made with Fehling's after they have been ground up, mixed with water, filtered through a cheese cloth and the filtrate tested for reducing sugar. Corn and oats were ground up in our experiments, mixed with water and the filtrate tested for reducing sugar. Sugar in from 0.000 grams in a sample of corn meal to 0.10 grams in samples of corn and oats are shown in table 10. Some of the samples were digested two hours, some were not digested at all. With one exception all samples showed a reducing sugar. The digestive action of mixed mouth saliva and parotid fistula saliva from the horse upon corn and oats is indicated in table 11. All of the samples show sugar. The average quantity does not appear to be higher than that from the grains alone as indicated in table 10.

One c. c. of parotid fistula saliva from the horse as shown in table 9 does not clear the starch solution. Reaction to N/20 iodine is G and the amount of sugar produced in any case is negligible. Parotid saliva in quantities larger than one c. c. and digested up to 24 hours changes the starch to erythrodextrin and produces sugar in an amount that can be measured by Benedict's reagent.

Details of an experiment in which extracts of the salivary and other glands from the mouth of the horse were used in conjunction with parotid fistula saliva to demonstrate the presence of an orokinase are given in table 12. Digestion was carried on for twenty-four hours. In none of the samples was the starch solution cleared. Reaction to N/20 iodine was G in all but three samples. Those were samples four and thirteen. The largest quantities of sugar are indicated in samples six and ten. The amount in each of these is 0.004 grams. The others show negligible quantities or none at all. Extracts from four different horses have been tested in this way.

We have had at our disposal only one horse with an esophageal fistula. The results in this case were disappointing. The action of mixed saliva from the fistula produced 0.004 grams of sugar. N/20 iodine gave reaction G. Oats that had been thoroughly masticated were obtained from the fistula, mixed with a small amount of water, filtered through cheese cloth and one c. c. of the filtrate tested for sugar. The one c. c. of filtrate gave 0.004 grams. A portion of the same bolus was allowed to stand for twenty-four hours and tested in like manner. The result was 0.003 grams of sugar. 0.2 HCL was added to another portion and at the end of twenty-four hours was tested. Two c. c. of the filtrate gave 0.002 grams of sugar. One c. c. of the mixed saliva obtained on the following day was digested for twenty-four hours with the corn starch solution. The starch was not cleared, N/20 iodine gave reaction S. The digestion produced 0.008 grams of sugar. Ground corn and oats were digested twenty-four hours with one c. c. of this same sample. The filtrate from the corn gave 0.000 grams of sugar. That from the oats gave 0.001 grams. This sample of mixed saliva did not reduce Benedict's reagent. The amount of digestion for twenty-four hours is slight. Any

comparison made between the material obtained from this fistula and the amylolytic power of human saliva would be overwhelmingly in favor of human saliva.

TABLE I

Action of Pilocarpine and Arecoline Upon 1 *Per Cent. Corn Starch. Time of Digestion Two Hours. * Indicates no Digestion.*

	Starch	N/20 I	Sugar grams
Pilocarpine crystals	0	G	.000
*Pilocarpine crystals			.000
*Strasenburgh hypodermic tablets			.023+
*Strasenburgh hypodermic tablets			.023+
Strasenburgh hypodermic, human saliva	0	R	.023+
Strasenburgh hypodermic, human saliva	0	R	.023+
Human saliva I	0	—	.023
Human saliva II	0	—	.023
Pilocarpine crystals	0	G	.000
*Pilocarpine crystals			.000
Pilocarpine crystals plus one c. c. parotid saliva horse	0	G	.000
Hypodermic tablets	0	G	.016
*Hypodermic tablets			.023
Pilocarpine crystals	0	G	.000
*Pilocarpine crystals			.000
Arecoline, hypodermic	0	G	.023+
*Arecoline, hypodermic			.023+
Pilocarpine, hypodermic	0	G	.018
*Pilocarpine, hypodermic			.019

TABLE II

Action of Two Drops Human Saliva, Dilution 1–50, *on* 1 *Per Cent. Corn Starch, Time of Digestion Two Hours.*

Starch	N/20 I	Sugar
0	G	.01 grams
0	S	.01 grams

Table II — *Continued*

Starch	N/20 I	Sugar
0	R	.005 grams
0	G	.001 grams
0	S	.014 grams
0	F	.003 grams
0	R	.013 grams
0	F	.004 grams
0	R	.009 grams
0	F	.002 grams
0	S	.011 grams
0	F	.006 grams
0	R	.006 grams
0	R	.009 grams
0	S	.009 grams
0	R	.016 grams
0	F	.009 grams
0	F	.007 grams
0	R	.009 grams
0	F	.000 grams
0	R	.010 grams
0	R	.005 grams
0	G	.007 grams
0	R	.017 grams
0	G	.004 grams
0	R	.013 grams
0	R	.016 grams
0	R	.032 grams
*0	G	.005 grams
0	R	.013 grams
0	R	.016 grams
0	F	.012 grams
0	F	.012 grams
0	F	.004 grams
0	R	.016 grams
0	F	.010 grams

* Dilution 1–100, 2 drops used.

TABLE III

Action of Two Drops Human Saliva, 1–50, *Plus Mixed Horse Saliva From the Mouth on Starch. Time of Digestion Two Hours.*

Starch	N/20 I	Sugar
0	G	.014 grams
0	S	.011 grams
0	G	.008 grams
0	G	.009 grams
0	V. S.	.006 grams
0	G	.011 grams
0	S	.011 grams
0	S	.009 grams
0	F	.010 grams
0	S	.009 grams
0	R	.014 grams
0	G	.003 grams
0	F	.003 grams
0	F	.006 grams
0	G	.004 grams
0	F	.003 grams
0	F	.005 grams
0	F	.001 grams
0	G	.000 grams
0	R	.013 grams
0	R	.017 grams
0	F	.001 grams
0	R	.010 grams
0	R	.023 grams

TABLE IV

Action of Two Drops Human Saliva, 1–50, *Plus* 1 *c. c. Parotid Fistula Saliva From the Horse on* 1 *Per Cent. Starch. Time of Digestion Two Hours.*

Starch	N/20 I	Sugar
0	G	.002 grams
0	G	.008 grams
0	G	.007 grams

Table IV — *Continued*

Starch	N/20 I	Sugar
0	S	.003 grams
0	S	.008 grams
0	G	.004 grams
0	F	.005 grams
0	G	.004 grams
0	R	.014 grams
0	F	.008 grams
0	F	.007 grams
0	G	.004 grams
0	R	.016 grams
0	R	.023 grams
0	R	.032 grams
0	R	.030 grams
0	R	.013 grams

Table V

Action of 1 *c. c. Mixed Human Saliva on* 1 *Per cent. Starch. Time of Digestion Two Hours.*

Starch	N/20 I	Sugar
0	V. S	.032 grams
0	R	.022 grams
0	—	.032 grams
0	—	.019 grams
0	—	.020 grams
0	—	.018 grams
0	—	.020 grams
0	—	.019 grams
0	—	.021 grams
0	—	.021 grams
0	—	.023 grams
0	—	.023 grams
*0	—	.023+ grams
0	—	.021 grams
0	—	.022 grams
0	—	.017 grams

* Same result from 12 different samples not included in the table.

TABLE VI

Action of 1 *c. c. Mixed Horse Saliva From the Mouth on Starch Solution. Time of Digestion Two Hours.*

Starch	N/20 I	Sugar
0	G	.001 grams
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	G	.001 grams
0	G	.003 grams
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	F	.000 grams
0	G	.001 grams
0	G	.000 grams
*0	G	.009 grams
*0	G	.004 grams

TABLE VII

Action of Two Drops of Mixed Horse Saliva From the Mouth, 1–10, *on Starch Solution. Time of Digestion Two Hours.*

Starch	N/20 I	Sugar
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	G	.006 grams
0	G	.003 grams
0	G	.000 grams
0	G	.003 grams
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams

* Sugar estimated with Fehling's solution.

Table VII — *Continued*

Starch	N/20 I	Sugar
0	G	.000 grams
0	G	.007 grams
0	R	.011 grams
0	G	.001 grams
*0	G	.000 grams

Table VIII

Action of Mixed Horse Saliva From the Mouth, Two Drops in a Dilution 1–10, *Plus* 1 *c. c. Parotid Saliva on Starch. Time of Digestion Two Hours.*

Starch	N/20 I	Sugar
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	G	.004 grams
0	G	.000 grams
0	G	.000 grams
0	G	.000 grams
0	R	.003 grams
0	G	.000 grams
0	G	.000 grams
†0	F	.004 grams
†0	G	.007 grams
0	G	.000 grams
0	G	.000 grams

Table IX

Action 1 *c. c. Parotid Fistula Saliva From the Horse on Starch. Time of Digestion Two Hours.*

Starch	N/20 I	Sugar
0	S	.000 grams
0	G	.000 grams

* Other samples gave the same reaction. Not included in the table.
† These samples were digested 26 hours.

TABLE IX — *Continued*

Starch	N/20 I	Sugar
0	G	.000 grams
0	G	.000 grams
0	G	.002 grams
0	G	.000 grams
0	G	.001 grams
0	G	.000 grams
*0	R	.000 grams
0	G	.000 grams
0	R	.002 grams

TABLE X

*Showing the Amount of Reducing Sugar in Ground Corn and Oats. Time of Digestion Two Hours. * Indicates No Digestion. Samples Were Tested Right After Mixing with Water. Sugar Reported is the Quantity in the Whole Filtrate.*

Grain	Amount	Amount H_2O	Sugar
Corn	20 grams	100 c. c.	.04 grams
Oats	20 grams	100 c. c.	.06 grams
Corn	10 grams	50 c. c.	.08 grams
C. meal	2 grams	25 c. c.	.02 grams
Oats	2 grams	25 c. c.	.04 grams
C. meal	2 grams	25 c. c.	.000 grams
C. meal	2 grams	25 c. c.	.025 grams
Oats	2 grams	25 c. c.	.025 grams
Corn	2 grams	25 c. c.	.086 grams
Oats	5 grams	25 c. c.	.085 grams
*Corn	5 grams	25 c. c.	.019 grams
*Oats	5 grams	25 c. c.	.012 grams
*Corn	5 grams	25 c. c.	.015 grams
*Oats	5 grams	25 c. c.	.017 grams
Oats	5 grams	25 c. c.	.15 grams
Corn	5 grams	25 c. c.	.10 grams
*Oats	5 grams	25 c. c.	.10 grams

* Ten c. c. of saliva digested.

Table X — *Continued*

Grain	Amount	Amount H_2O	Sugar
*Corn	5 grams	25 c. c.	.09 grams
Oats	5 grams	25 c. c.	.029 grams
C. meal	5 grams	25 c. c.	.019 grams

Table XI

Action of Saliva of the Horse on Ground Grains. Time of Digestion Two Hours. Five Grams of Grain to 25 c. c. of Water in Each Sample.

Grain Saliva	Amount	Sugar
Corn — Mixed	5 c. c	.055 grams
Corn — Parotid	1 c. c.	.017 grams
Oats — Parotid	2 c. c.	.035 grams
C. meal — Mixed	1 c. c.	.050 grams
C. meal — Parotid	1 c. c.	.032 grams
Corn —Mixed	1 c. c.	.067 grams
Corn — Mixed	1 c. c.	.035 grams
Oats — Mixed	1 c. c.	.050 grams
Corn — Mixed	1 c. c.	.043 grams
Oats — Mixed	1 c. c.	.057 grams
Corn — Mixed	1 c. c.	.027 grams
Corn — Mixed	1 c. c.	.045 grams
Corn — Mixed	1 c. c.	.033 grams
Oats — Mixed	1 c. c.	.046 grams
Oats — Mixed	1 c. c.	.045 grams
Corn meal — Mixed	1 c. c.	.075 grams
Corn meal — Mixed	1 c. c.	.090 grams
Corn meal — Parotid	10 c. c.	.058 grams

Table XII

Action of Various Gland Extracts on Starch. 0.5 *c. c. of Extract Used. Where Parotid Saliva is Used* 1 *c. c. is the Amount. Time of Digestion* 24 *Hours.*

Material	Starch	N/20 I	Sugar grams
Buccal gland	0	G	.000
Buccal gland plus parotid saliva	0	G	.000

TABLE XII — *Continued*

Material	Starch	N/20 I	Sugar grams
Parotid saliva	0	G	.000
Parotid saliva, 10 c. c.	0	R	.000
*Buccal gland			.000
Buccal gland plus parotid ext.	0	G	.004
Buccal gland plus sub. max. ext.	0	G	.002
Buccal gland plus sub. ling. ext.	0	G	.000
Lingual glands	0	G	.000
Lingual glands plus parotid saliva.	0	G	.004
*Lingual glands			.000
Lingual glands plus parotid ext.	0	G	.001
Lingual glands plus sub. max. ext.	0	F	.001
Lingual glands plus sub. ling. ext.	0	G	.002
Mucosa, mouth	0	G	.001
Mucosa plus parotid saliva	0	G	.002
*Mucosa			.000
Muccosa plus parotid ext.	0	G	.000
Mucosa plus sub. max. ext.	0	G	.001
Mucosa plus sub. ling. ext.	0	G	.001
Parotid ext.	0	G	.002
Sub. max. ext.	0	G	.001

TABLE XIII

Material from Esophageal Fistula.

	Starch	N/20 I	Sugar grams
1. Starch plus mixed saliva	0	G	.004
2. Starch plus ½ c. c. mixed saliva	0	S	.003
3. Oats from fistula, 1 c. c. of filtrate			.004
4. Oats from fistula, 1 c. c. of filtrate, 24 hours			.003
5. Oats from fistula, 2 c. c. of filtrate, 0.2 HCL			.002
6. Starch plus mixed saliva, 24 hours digestion	0	S	.008

* Indicates no digestion.

Table XIII — *Continued*

Material	Starch N/20 I	Sugar grams
7. Ground corn 2 grams, 25 c. c. H20 plus 1 c. c. mixed saliva.....		.000
8. Oats ground 2 grams, 25 c. c. H20 plus 1 c. c. mixed saliva.....		.001
9. 1 c. c. mixed saliva, not digested...		.000

Discussion

The samples of pilocarpine hydrochloride in crystal form did not digest starch. The hypodermic tablets of neither pilocarpine or arecoline show digestive power. They do contain a reducing substance which is easily measured. When 0.5 grain tablets of either were dissolved in 10 c. c. of water and 2 c. c. of the solution tested that small quantity completely reduced the amount of the reagent used. There is no evidence in our experiments that the pilocarpine in either form digests starch.

Orokinase in the saliva of man is not shown to exist according to our results. Two drops of human saliva in the dilution 1–30 when acting on the starch solution for two hours show considerable digestive power. The amount of sugar produced by most of the samples would give a heavy reduction with Fehling's solution. One sample in a dilution of 1–100 showed a marked activity. A comparison of the action of human saliva diluted 1–50 with that of saliva in the same dilution plus mixed horse saliva or human saliva plus parotid fistula saliva from the horse does not reveal any marked increase in digestion. None of the samples in Tables 3 and 4 show any more than is shown in Table 2. From our data we cannot claim the presence of an orokinase in the saliva of man that activated the mixed mouth saliva or the parotid fistula saliva of the horse.

A comparison of the action of human and mixed horse saliva upon a cooked starch solution is very much in favor of the power of the human saliva. The same thing holds true when parotid fistula saliva is considered. Human saliva invariably had a much stronger action on the raw starch of corn and oats than either sample from the horse.

Two drops of mixed horse saliva from the mouth diluted 1–10 show fully as much action on starch as the combination of that dilution with parotid fistula saliva.

Repeated tests with corn or oats ground in a food chopper, mixed with water and filtered through cheese cloth give evidence of reducing sugar. The quantity of sugar varies from 0.000 grams in a sample of corn meal to 0.15 grams in a sample of ground oats. The amount of sugar obtained from either of these grains after having been digested with either mixed or parotid fistula saliva from the horse does not average higher than the grains alone when mixed with water. There is evidence in the literature already quoted of sugar in these grains and of the presence of a diastase that may be activated by grinding them. The diastase is also said to be active in the grains without any mechanical change in them.

Glycerine extracts of the mucosa of the mouth, of the buccal glands, and of the salivary glands from four different horses have not shown any marked activating influence when digested with parotid fistula saliva or indifferent combinations with each other. We have too few of this series to come to a definite conclusion. Some of the digested products if tested with Fehling's solution alone would show a marked reduction. Orokinase cannot be demonstrated by the use of that reagent alone. We have a large number of samples in the different phases of the work that would give some considerable reduction. When the quantity of sugar was measured it was found that it was not appreciably greater in the material in which no enzyme was supposed to have been present. Two drops of human saliva diluted 1–50 gave frequent erythrodextrin reactions when digestion was carried two hours. None of the samples tabulated in Table XII did save one and 10 c. c. of parotid fistula saliva was used in that. No sugar could be measured from this sample. Parotid saliva in quantities greater than 1 c. c. when digestion is carried up to 24 hours frequently give the erythrodextrin reaction and measurable quantities of sugar. The same thing has occurred with the mixed saliva and the various extracts that have been made. This seems to be in accord with the results obtained by Seymour (2).

The amount of sugar measured in these cases has been a disappointment to us. We feel that an enzyme in the mouth secretions activating the salivary secretions would lead to the produc-

tion of measurably more sugar than that produced from the secretions or extracts used alone. That has not been the case in our experiments.

Our one esophageal fistula has not given the results hoped for. The amount of sugar produced in any experiment with the mixed saliva from that source has been small. In no wise has it been comparable with the action of human saliva on either cooked or raw starch.

Neither oats or corn having been thoroughly masticated and passed through the fistula have given more sugar than we have obtained from the ground grains in water. Mixed saliva from this fistula did not show digestive action on these grains. The mixed saliva did not show any reducing power. Mixed saliva acting on cooked starch for 24 hours produced 0.008 grams of sugar. The human saliva in the dilution used produced as much or more when digestion was carried on for only two hours.

Conclusions

1. Pilocarpine hydrochloride does not digest starch. Hypodermic tablets of both pilocarpine and arecoline contain a reducing substance in comparatively large quantities but do not in themselves digest starch.

2. Two drops of human saliva diluted 1–50 carry 5 c. c. of a 1 per cent starch solution to the erythrodextrin stage in a large number of cases. A measurable amount of sugar is produced as a result of that digestion.

3. Human saliva in such a dilution does not activate either mixed or parotid fistula saliva from the horse.

4. Mixed human saliva digests cooked starch much more readily than either mixed or parotid fistula saliva of the horse.

5. Two drops of mixed mouth saliva from the horse diluted 1–10 does not activate parotid fistula saliva from that animal. It does not show any appreciable digestive power when used alone in that dilution.

6. The filtrate from a solution of ground corn or oats contains a reducing sugar. The quantity of sugar does not show an average increase when the grains are digested with either mixed or parotid fistula saliva from the horse. Mixed human saliva does digest them under the same conditions.

7. Extracts from the glands and mucosa of the mouth have failed to activate parotid saliva or extracts of the salivary glands of four different horses.

8. Corn and oats passed through an esophageal fistula show no more reducing sugar than the ground grains themselves. Mixed saliva from the esophagus has not shown any marked potency.

9. The glands of the mouth as well as the salivary glands produce a small amount of enzyme that will digest starch within a 24-hour period.

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TUBERCULOSIS IN EQUINES

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Equine tuberculosis is a rare disease. It is indeed, but perhaps not as rare as some of our own individual experiences might lead us to believe. A careful review of the literature will bring to light the fact that quite a number of cases have been recorded in which the diagnosis was confirmed by microscopical examination. Many cases have occurred spontaneously and also the disease has been produced by inoculation. This establishes the susceptibility of the horse to tuberculosis. It therefore behooves the veterinarian as the guardian of the health of the live stock of this country to be on the lookout for tuberculosis in horses as well as in bovines.

The fact that the disease is not met with in the daily routine is hardly a valid excuse for failure in its diagnosis if encountered. Further, it may be stated that it is the unusual cases that really test ability. It is by the manner in which we handle these cases that we may judge ourselves and that we are largely judged by our fellow practitioners.

From a monetary standpoint, the losses occuring from equine tuberculosis in Europe are not very great although they can hardly be ignored. Also, if we accept the number of cases recorded in this country as an index to the actual number occurring, the loss would be small. It is probable that there are cases which are not diagnosed and also those which are diagnosed but not reported. It is true, however, that each individual animal has its place and a specific function to perform. Its death represents a shortage in labor and the loss of its actual value to the owner as well as a direct loss to the government. The prevention of the exposure of these animals to the disease is therefore a distinct service to the owner as well as to the nation.

A brief description of the disease as compiled from case reports and text books follows.

History.—According to Hoare (21), tuberculosis in the horse was probably first recognized in Europe by Nocard and others

in 1878. From that time until 1896, the number of cases published would approximate one hundred. M'Fadyean (24) should be largely credited for this as he called the attention of the profession to the presence of the disease in equines. He also recorded the first two cases of the malady in England after the discovery of the tubercle bacillus. These cases were reported in Volume I of the Journal of Comparative Pathology and Therapeutics, 1888. From that date to 1892, twenty-eight cases were recorded in that journal. Until a comparatively recent date the disease was often mistaken for a condition termed "lymphadenoma" especially in the spleen. M'Fadyean demonstrated that this so-called lymphadenoma was tuberculosis.

In Robertson's Equine Medicine published in 1892 we find it stated that the situation of the localized tubercles are chiefly in the gland structures of the abdomen. In describing lymphadenoma of the spleen, he admitted that in three or four cases the tubercle organisms were found in great numbers. He also stated that joint ill was of tubercular character.

Occurrence.— Tuberculosis in horses has been reported from nearly every country in Europe. In England alone the Journal of Comparative Pathology and Therapeutics and the Veterinary Record have contained descriptions of over fifty cases.

In America we have found the reports of only two cases. One of these was recorded by Dr. Shigley (35) of North Dakota. The other was noted by Dr. Fink (17) of New York. The author had the privilege of confirming this last diagnosis in the laboratory and it is being described as case No. 1 in this article.

In regard to the occurrence of tuberculosis in horses of different ages, the following 43 cases may be of interest. They were: 1 yearling, 7 four year olds, 4 five year olds, 3 eight year olds, 4 ten year olds, 2 eleven year olds, 1 twelve year old, 1 thirteen year old, 1 sixteen year old, 1 eighteen year old, and 6 in which the age was not stated.

As to breed, the highly bred horses were in the minority. Thus, 18 were cart horses, 10 harness horses, 2 well bred animals, 1 troop horse, 1 Belgian stallion, 2 ponies, 1 cob, 1 hackney and 7 in which the breed was not given.

Source of Infection.— In a number of cases the history indicates that the animals were reared on cow's milk in consequence of having lost their dams shortly after birth. It is also a well known fact that in Europe and to some extent in America weak foals or horses suffering from debilitating diseases are fed on cow's milk; if the latter contains the organism the source of infection is reasonably clear.

Davis (12) recorded 4 cases which occurred on one farm. They were a 4 year old hackney mare, a yearling filly, the daughter of the mare, a 6 year old pony and a mare which was the grand-dam of the filly. The yearling filly was reared on cow's milk and was in association with the pony.

Nocard believed that the abdominal type of the disease was due to an infection from an avian source while the pulmonary depended upon either the bovine or human organism. M'Fadyean did not agree with this view insofar as the avian source was concerned. The Royal Commission on Tuberculosis investigated 5 cases of the disease and concluded that the organisms recovered by them resembled more closely those of the bovine than either the human or avian types. Shigley states, however, that the case described by him contained the human type.

Channels of Infection.—A peculiar feature noted in tuberculosis in the horse as compared with the malady in cattle is that the lesions are a little differently distributed. The mesenteric lymph glands and the spleen are more often involved and are generally the seats of the primary lesions. This would point rather strongly to the intestines as being the path by which the organisms gained entrance to the tissues.

Smith (36) inoculated, by means of a hypodermic syringe, tubercular material from a human lung into the trachea of a horse but was unable to produce the disease.

Symptoms.— The most constant symptom is a history of gradual emaciation extending over a period of several months. In a few cases recorded the loss of condition followed closely upon an attack of influenza. The temperature is generally raised slightly. It may be 101° or 102° F. In a few cases it has been recorded as high as 103° F. and is subject to marked fluctuations. The appetite is often variable but may be good until the disease is

well advanced. In other cases a marked anorexia has been noted early. The visible mucous membranes are sometimes anemic. Swelling of the limbs may occur.

When the lungs are involved a cough may or may not be observed. The respiration often simulates that of wind broken animals. In case of abdominal lesions colicky pains are noted especially after feeding. In involvement of the glands of the head or neck, deglutition and respiration may be interfered with. When the vertebrae are diseased the symptoms correspond to the part of the column affected.

Post Mortem Findings.—An interesting feature of this disease is the frequency with which the vertebrae are involved. The lesions here consist of an ostitis and a periostitis. The cervical vertebrae seem to be the most often affected. Cases are described in which only one or two are involved while in others they may all show morbid changes. The lesions may be irregular scaly exostoses confined to the exterior of the bones, giving them a spongy appearance. Other cases may show the articular surfaces and the spinal canal involved. In still others complete ankylosis may be noted.

Tuberculosis in the lungs most often takes the miliary form. Sometimes the so-called sarcomatous appearing nodules are observed. These are firm in consistency and generally do not show a necrotic centre. Again lesions apparently identical with the caseous nodules found in cattle have been noted. Calcified and well encapsulated lesions are also not uncommon. DeVine (13) recorded a case in which the lungs were much enlarged. They weighed 77 pounds.

The bronchial lymph glands have often been enlarged, sometimes greatly so and caseated. They have on several occasions been practically normal in case of marked involvement of the lungs.

The mediastinal lymph glands are occasionally affected. In one case a large growth was observed in the anterior mediastinum. On section it was found to be caseous and contained some calcification. The prepectoral, prescapular, inguinal and other superficial lymph glands may show lesions. The submaillary and pharyngeal lymph glands have also been reported as showing lesions in several cases.

The pleura was affected in 7 cases out of a total of 54. It is not generally as extensively involved as in cattle but its lesions resemble the latter very closely.

The liver is occasionally the seat of the disease. When affected it may present only a few nodules and those of small size. In one case the lesions were described as pale yellow tumors.

The spleen seems to be the most often involved. The lesions consist of nodular growths of various sizes. Some are firm in consistency and present on section a yellowish white mass with or without caseation. Again they may contain caseo-purulent material. The whole organ may be greatly enlarged weighing as much as 16 and even 23 pounds. The splenic lymph glands are sometimes involved and may or may not show caseation.

The mesenteric lymph glands are usually affected. They generally show a marked enlargement with extensive caseation. It is not unusual to find them in masses in which condition they have grown by enlargement and becoming confluent with their neighbors.

The peritoneum has been found to be diseased several times. In certain cases it was covered with small tubercles (Pearl disease). In others it showed the nodules less numerous. In two cases the peritoneal surface of the diaphragm was the extent of the lesions.

The kidneys are only rarely tubercular. Out of 54 cases, only 1 showed tuberculosis in these organs. They are, however, generally much enlarged, weighing as high as 6 pounds.

Lesions of the stomach and intestines are also rare. One case of involvement of the serosa of the stomach, 1 of the serosa of the large intestine, 3 of ulcers of the small intestine and 1 of ulcers of the large intestine have been reported. Hutyra and Marek (22) state that the nasal mucous membrane is sometimes involved as well as the skin and subcutem.

The following table gives the organs affected in 54 cases of the disease. In the perusal of these figures a slight discrepancy will be noted. This is due to the fact that the percentages have been carried out only one decimal place.

Organs Affected in Fifty-Four Tubercular Horses

	Number of times	Percentage of 54	Percentage of 175
Submaxillary lymph gland	3	5.5	1.7
Bronchial lymph gland	8	14.8	4.5
Mediastinal lymph gland	4	7.4	2.2
Mesenteric lymph gland	32	59.2	18.3
Sublumbar lymph gland	4	7.4	2.2
Inguinal lymph gland	1	1.8	.5
Cervical vertebrae	5	9.2	2.8
Lungs	35	64.	20.
Pleura	7	12.7	4.
Heart	3	5.5	1.7
Diaphragm	8	14.8	4.5
Spleen	38	70.3	21.7
Liver	7	12.7	4.
Peritoneum	8	14.8	4.5
Stomach (serosa)	1	1.8	.5
Small intestine (ulcers)	3	5.5	1.7
Large intestine (serosa)	1	1.8	.5
Large intestine (ulcers)	2	3.7	1.1
Kidney	1	1.8	.5
Ovary	1	1.8	.5
Testicle	1	1.8	.5
Muscle	1	1.8	.5

Course.— The course is variable. The disease may extend over a period of but a few weeks as in one case reported, or it may cover a year, 2 years or longer. In the great majority of animals affected, a slowly progressive emaciation is noted. This either ends in death from exhaustion or the animal becomes so debilitated that it has to be destroyed. One case, however, has been reported in which emaciation was not marked. We have practically no data in regard to the recovery of animals.

Diagnosis.— In animals showing well marked emaciation and in the absence of other apparent causes, tuberculosis may be suspected especially if any of the before mentioned symptoms are present. The tuberculin test should by all means be used when possible as there are numerous cases reported in which a

typical reaction has been obtained. A few cases have been described in which the reaction was not obtained. The technic of the test should be carried out exactly as in the bovine. The same dose may be used but should be varied with the size of the animal as for example a percheron might be given four cubic centimeters of tuberculin while a small pony would require but one cubic centimeter.

A peculiarity in the tuberculin reaction in equines is that a local reaction is practically always obtained. It consists of a firm swelling at the seat of injection. It may be as large as a walnut or even a saucer and larger. It disappears in from eighteen to forty-eight hours.

At autopsy, the lesions of the lungs are generally miliary which are easily differentiated from most cases of glanders. The changes in the lymph glands and especially the spleen may give some trouble in the gross examination but microscopical and bacteriological findings will identify them.

Case No. 1

The history and autopsy of this case were reported by Dr. J. W. Fink in the American Journal of Veterinary Medicine 1916, Volume II, page 143. They are in substance as follows: "About a year ago a very aged horse came to my observation for treatment, with the history that the animal had not been doing well and seemed to be running down as the owner described it. After carefully examining the patient, I was unable to arrive at any definite conclusion or find any evidence of clinical disturbance other than what may have been mistaken for pulmonary emphysema or heaves. Temperature 106° F. I advised the owner to relieve the horse of any hard work for a while and feed him well and allow him the freedom of the paddock.

"Four months later I had occasion to visit the farm and while there examined the horse as the owner thought the animal appeared to be just about the same in spite of the better care and feeding he was receiving. At this time the horse had a temperature of 101° F. and when moved about, would emit a slight cough not unlike a 'heavey cough', though not characteristic nor was the respiration characteristic of emphysema. The

animal continued to fail for the next six months and was finally found dead one morning in the stall.

"The post mortem examination revealed lesions so similar to bovine tuberculosis that I removed sections for histological and bacteriological examinations to verify the diagnosis. The lungs were considerably enlarged, there being masses of tumor-like formations the size of walnuts throughout both lungs, each tumor being surrounded by complete or incomplete fibrous connective tissue capsules — the centres of many calcareous and a few caseo-purulent. The mediastinal and bronchial lymph glands were enormously enlarged and quite hardened by fibrous tumor formations with small caseous and calcareous centres. The functional tissue of the spleen was almost completely obliterated by the presence of tumors varying in size from that of a walnut to a lemon. Many were calcareous, a few caseous and all surrounded by thickened fibrous connective tissue walls almost tendinous. The liver contained only two lesions of a similar character, of about the same consistency, close together and on the under side. The lesions throughout resembled somewhat the tumors described as lymphadenomata, altogether varied in size from that of a cent to an orange, in a few instances.

"It might be interesting to note that this animal had always been stabled where the stalls opened into the cow barn and pastured with cattle that had never been tuberculin tested. The attendants and owner as well showed physical evidence of tuberculosis."

The tissues received from Dr. Fink for our examination were the right lung, mediastinal lymph gland and a portion of the spleen.

Description of Organs.— The right lung was somewhat enlarged with all of its borders more or less rounded. The pleura was much thickened and was wrinkled in places. It was yellowish white in color and was firm on pressure. In numerous places were noted many small and rather delicate fibrous tufts giving it a roughened appearance. It was otherwise smooth and glistening. In a few places there were irregular rounded elevations projecting above the surface from a small distance to 2½ cm. in diameter. These elevations were lighter in color than the surrounding pleura.

which was due to the tubercles beneath. There was also present a large scar about 3 cm. long by 2 cm. wide, the centre of which was indented about 1 cm. This was of long standing and indicated that a rather large tubercle had some time eaten its way through the pleura and ruptured on the surface of the organ.

On section it was found that at least one-half of the lung tissue was involved. There were scattered lobules near the surface of the organ which appeared to be practically normal. The central portion of the organ measuring about 15 x 20 cm. was entirely changed, there being no functionating tissue left, only the larger bronchi remaining, some of which presented partially broken down walls. The tubercular tissue seemed to be one large irregular dense mass of a light yellowish white color, containing several irregular darker colored caseated areas varying in size from just visible to a centimeter in diameter. A few of these areas contained small amounts of a finely granular calcification. Around the borders of the large central mass were numerous irregularly rounded independent nodules measuring up to 1 cm. in diameter, situated in the relatively normal lung tissue. These nodules generally contained no calcification or caseation. The tissue was dense and firm throughout. In most cases there were no sharp lines of demarcation between the normal and the affected tissue nor were there evidences of encapsulation. A few lobules of the lung near the surface of the organ showed a small amount of emphysema.

The mediastinal lymph gland was about 7 cm. x 6 cm. x 4 cm. It was greyish white in color and was of irregular shape. On section it was found that the capsule and the larger trabeculae were practically all of the original tissue remaining. The medulla was simply an irregular mass of pinkish yellow color containing areas of caseation which showed small quantities of a finely granular calcification.

The portion of the spleen showed that this organ was much enlarged. The capsule was thickened and in places small fibrous tufts were present. The organ was made up of masses of rounded tubercles which were light in color and raised 2 to 3 cm. above its surface.

On section is seemed to be composed almost entirely of these

tubercles, many of which had grown until they had reached their neighbors and had become confluent with them thus forming large irregular masses. There were sharp lines of demarcation between the normal and the affected tissues in a few places and also distinct capsules surrounded a few of the nodules.

The individual tubercles were composed of the same light yellow tissue described in the lung and lymph gland. Scattered irregularly through this tissue were numerous small irregular areas, varying in size up to 1 cm. in diameter, of caseation some of which contained a small amount of calcified granules. Rarely a nodule was noted which contained a necrotic centre in which softening and liquefaction had started to take place.

Smears were prepared from the diseased tissues of each of the organs received and were stained for the tubercle organism. On examination each of these revealed enormous numbers of *Bact. tuberculosis.* Pieces were then taken from several different parts of each of the organs for histological examination. These were fixed, dehydrated, cleared and sectioned in the usual manner. They were stained with the Ziehl Neelsen stain and with hematoxylin and eosin.

Histological Examination.— The sections of lung, lymph gland and spleen upon examination showed numerous acid fast bacteria identical morphologically with *Bacterium tuberculosis.*

The specimens stained with hematoxylin and eosin were next examined.

In the lung there was practically no normal tissue found. The parts farthest away from the larger nodules and away from the immediate vicinity of the smaller ones showed a broncho-pneumonia. The bronchial epithelium was pretty largely desquammated and lay free within the bronchi together with much fibrin and some leucocytes. In the immediate vicinity of the bronchi the walls of the air sacs were enormously congested and the sacs themselves were filled with leucocytes and fibrin. Still farther away and only rarely were observed small areas in which the only changes noted were congestion and the presence of leucocytes in the air sac walls. The whole organ presented an active congestion. The pleura was thickened which was due to some connective tissue proliferation but mainly to an oedema. The inter-

lobular connective tissue was much thickened due to the same causes.

The tubercular nodules were scattered irregularly and very thickly throughout the lung tissue. They were found to be more numerous in some places than in others. They varied in size from minute points containing only a clump of lymphocytes to enormous areas in which numerous nodules had become confluent.

The smaller nodules were generally located in a hepatized area. Their structure was very similar to the miliary tubercles of bovines but the different zones were not as clearly discernible. The central portions were composed of necrotic material which usually contained many lymphocytes. A zone of giant and epithelioid cells could not be made out. In its place was found a comparatively wide area containing many lymphocytes in a rather delicate connective tissue stroma. This zone seemed to radiate out into the lung tissue. In this zone giant cells were sometimes noted although very rarely more than one and often not even one in a nodule. The central portions of the smaller tubercles did not contain calcification and only a few of them showed connective tissue capsules.

The large tubercular areas were lobulated similar to the lung tissue with a dense fibrous interlobular connective tissue. Within the lobule were found from one to five or six rather small necrotic areas which might or might not contain calcification. The remaining tissue seemed to be lymphocytes more or less thickly crowded into the meshes of the connective tissue stroma. Very few giant cells were noted in these lesions.

The mediastinal lymph gland was also badly affected. The capsule and the trabeculae were much thickened, which seemed to be entirely due to connective tissue proliferation. This thickening materially restricted the area occupied by the lymphoid tissue. The medulla was almost entirely tubercular. It contained numerous necrotic foci varying greatly in size. Some were minute while others occupied larger areas. These were rarely calcified. Around them were found sparsely scattered lymphoid cells held in a heavy connective tissue stroma. There were but few giant cells observed and these were present in the areas surrounding the smaller necrotic centres. They were not noted in the lesions of long standing. The whole gland showed some congestion.

The spleen was literally filled with tubercular nodules. These varied in size from minute up to macroscopic lesions which could be measured in centimeters. Their structure was similar to that found in the lymph gland. The necrotic centres were generally small. They sometimes contained calcification although not always. A few of these centres showed liquefaction necrosis. Around them was found the heavy connective tissue stroma containing numerous lymphocytes. This zone only rarely contained giant cells. In some of the nodules the edges radiated out into the splenic tissue or into a similar zone surrounding a neighboring necrotic centre while in others the edges were more or less sharply defined and in still others there was evidence of a connective tissue capsule.

The splenic capsule was thickened by oedema and connective tissue proliferation. The trabeculae were larger than normal which was due to an increase in the connective tissue alone. The splenic pulp still remaining showed a marked congestion. It otherwise appeared to be normal.

Case No. 2

We have unfortunately been unable to obtain the history or the complete post mortem notes from this animal. The veterinarian in charge of the case stated that he received a telephone call from a client requesting him to come and make a post mortem examination on a horse that had just died.

Upon opening the animal it was found that the lungs were practically filled with lesions which appeared to be glanders nodules. The spleen was also in the same condition. Whether or not other lesions were present we are unable to state. The diagnosis of glanders was, however, tentatively made and the lung and spleen were shipped to this laboratory for confirmation of the field diagnosis.

The specimens were received in a good state of preservation and are described as follows:

The left lung was much enlarged and all of its borders were rounded. The pleura was thickened and of a bluish white color over the lung tissue and a yellowish white color over the tubercular tissue. It was smooth and had a glistening appearance gen-

erally but in places it was roughened by fibrous tufts. The pleura was slightly raised in several places by small light colored tubercles which varied in size up to 1 cm. in diameter. There was one place where the pleura had ruptured exposing tubercular tissue beneath. There were also present a few nodules about 5 mm. in diameter which appeared to be entirely within the pleura.

On section it was found that practically the whole lobe with the exception of about 10 cm. at the apex was involved. This portion measured 16 x 17 x 15 cm. and appeared to be entirely tubercular. The only normal tissues remaining (macroscopically) were some of the larger bronchi and the interlobular connective tissue. This was much thickened measuring 2 mm. in some places and was somewhat oedematous. The intralobular tissue was firm and had the same characteristic, light yellow color, as described in the affected organs under case No. 1. Scattered irregularly through these lobules were numerous areas of calcification varying in size up to 1 cm. in diameter. This mass seemed to have started as small miliary tubercles in the different lobules which had grown until they had become confluent with their neighbors and only being held in check by the interlobular tissue. In some of the lobules several independent tubercles were noted with calcified centres. In the middle of the lobe was a large cavity about 7 cm. deep and 4 cm. wide in its widest place. There were several other cavities present generally situated in the centres of the lobules and varying in size up to 1 cm.

There was a sharp line of demarcation between the tubercular mass and the lung tissue at the apex of the lobe. The boundary line was made up entirely by the interlobular connective tissue.

The lung tissue at the apex of the lobe also contained several tubercular nodules varying in size up to 2 cm. in diameter. Some of these had started to show cavitation. The lung tissue proper was somewhat denser than normal which was probably due to pressure and was slightly congested. The interlobular tissue here had proliferated much and showed oedema. It was 3 mm. thick in places distinctly marking off the different lobules.

The spleen was of normal size. The capsule was also normal with the exception of being slightly roughened in places due to

the presence of numerous small fibrous tufts. Its surface was raised fully a centimeter in places by nodules measuring from 1½ to 3 cm. in diameter. One portion was dotted with small irregular shaped nodules from 2 to 5 mm. in diameter.

On section the larger tubercles were found to be filled with dense yellowish white material similar to that heretofore described. Small amounts of calcification were present in these areas. Some of them were well encapsulated while others were not and presented no distinct line of demarcation between the splenic and the affected tissues. In the immediate vicinity of the nodules the pulp was slightly congested while away from them it appeared to be normal.

Smears were made from lesions of the lung and spleen and were stained after the Ziehl Neelsen method. They revealed enormous numbers of *Bact. tuberculosis*. Two guinea pigs were inoculated, one with material from the lung and the other with material from the spleen. Both died in about four weeks, each showing well marked tubercular lesions in which the organism was readily found. Pieces of tissue were also taken from both of these organs for microscopic examination as in the preceding case.

Histology.— The sections of these organs stained after the Ziehl Neelsen method showed numerous tubercle organisms. They were found almost entirely in the zone of lymphocytes but few being observed in the necrotic centres.

The lung was badly involved, there being practically no normal tissue left. Away from the tubercles the organ showed a slight bronchopneumonia. The interlobular connective tissue was greatly thickened which was principally due to oedema. The tubercular foci were scattered throughout the organ and varied in size from minute to enormous areas. The structure of the individual nodules was similar to that heretofore described. The necrotic centres were on the whole larger than those found in the previous case. They did not contain as many lymphocytes but more calcification. They also showed a greater tendency to undergo liquefaction which resulted in a good deal of cavitation. A few giant cells were noted in the zones of lymphocytes. Their borders were sometimes diffuse, spreading out into the lung tissue, while in others they were sharply defined. In some cases a heavy connective tissue capsule was noted.

The larger tubercular areas were nearly the same as those found in the other lung. They were limited in extent only by the interlobular connective tissue. A lobule generally contained several necrotic centres which might or might not show calcification. In these lesions cavitation was found to be not uncommon.

The spleen presented a marked congestion throughout. Otherwise it appeared to be normal with the exception of the tubercular lesions which were less numerous than in the previous case. The nodules were generally large in size only a few small ones being noted. Whenever present their structure showed but little not previously mentioned in the description of the other organs. The larger tubercular areas contained relatively small necrotic centres which appeared to be composed of caseation necrosis of rather firm consistency. But little calcification was found in these centres. The zone surrounding them was large and occupied the greater part of the lesions. Only a few giant cells were found in the nodules of this organ. The lymphoid zones generally radiated out into the splenic tissue with no sharp line of demarcation between the splenic and the tubercular tissue. There was a somewhat heavier congestion in the immediate vicinity of the tubercles than away from them. A few nodules showed encapsulation but no evidence of liquefaction was found in the necrotic centres.

Case No. 3

Again in this case we have been unable to obtain the history. The subject was an aged gelding weighing about 900 pounds. It had evidently outlived its usefulness and was brought to the college for destruction. It was extremely emaciated and was suffering from a chronic skin disease. The animal was destroyed with chloroform and the following lesions were found.

The lungs were comparatively normal with the exception of the apexes of both principal lobes. At the apex of the left lobe an area about 5 cm. by 3 cm. at its widest place was literally filled with tubercles. They varied in size from 1 mm. to 1 cm. in diameter. On section they were found to contain yellowish white caseous and sometimes calcified centres. They were in nearly every case surrounded by a dense connective tissue capsule about 1 mm. thick. The larger nodules were made up of smaller ones which had become confluent.

Dorsal to this area the nodules became less numerous until a distance of about 12 cm. from the apex was reached where no more were found. On the costal surface of this portion was a caseous mass (the largest tubercle found in the body) about 3½ cm. by 1 cm. It appeared to be situated in a thickening of the pleura. In the lung proper in the vicinity of this area were found several small nodules about 3 mm. in diameter. These were filled with caseous and sometimes calcareous material. The larger tubercles and several of the smaller ones showed areas of congestion immediately surrounding them. The lung showed some emphysema in places but otherwise appeared normal.

In the right lobe the lesions were present only in an area extending from the apex toward the trachea for a distance of about 16 cm. and about 12 cm. in its widest place. In this area were numerous tubercles varying in size from 1 mm. to about 3 cm. in diameter. On section these were found to be situated principally in the lung tissue and to contain caseous centres in which calcium salts were generally deposited. They were nearly always surrounded by a dense fibrous connective tissue capsule which varied in thickness with their size. The largest nodule in this lobe contained a greyish watery pus somewhat resembling mucus which under the microscope showed pus cells, lymphocytes, fibrin and cell detritis. Most of the tubercles were surrounded by an area of congestion. The lung was otherwise similar to the one previously described.

Smears were made from lesions of both lobes and were stained for the tubercle organism. Upon microscopical examination these bacteria were found but not as numerous as in the specimens previously examined. Pieces of tissue were next taken from tubercles of both lobes for sections.

Histology.— Examination of sections stained after the Ziehl Neelsen method showed *Bact. tuberculosis* but in relatively small numbers. They were noted only in the zone containing the lymphoid cells.

The examination of the sections stained after the hematoxylin and eosin method showed a slight congestion in places and rarely a little solidification. Some emphysema was noted but not enough to interfere with the functioning of the organ.

The tubercular lesions were similar to those of the other cases described but presented a few marked differences which would indicate a condition of longer standing as well as a disposition to encapsulation absorption and spontaneous recovery. The smaller nodules presented the usual necrotic centre but larger in proportion than in the other cases. They also contained more calcification and would not section without tearing in case the tissues were not previously decalcified. The zones of lymphoid cells in the connective tissue stroma which have been previously so well marked were almost or entirely absent. When present they consisted of a narrow border around the necrotic centre. The lymphocytes were few in number and the stroma was heavy and dense. Around this zone a fibrous connective tissue capsule was practically always found. It seemed to vary in thickness and to some extent in density with the size of the nodule. A zone of marked congestion was generally present in the lung immediately surrounding the capsule of the nodule.

The larger tubercles contained several necrotic areas which were often almost completely filled with calcified material. A few of these showed liquefaction with the presence of numerous pus cells and fibrin. The zone surrounding the necrotic areas was almost entirely devoid of lymphocytes. The connective tissue stroma was similar to that found in the smaller nodules but was somewhat heavier. No giant cells were noted in any of the larger nodules and only rarely in the smaller ones. Surrounding the masses of several necrotic centres and their contiguous zones was generally found a heavy connective tissue capsule. Outside of it the lung tissue usually presented a marked congestion in its immediate vicinity.

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Description of Plates

Plate No. I. Section of lung from Case No. 1. (One-half original size.) The lesions are generally neither surrounded by a connective tissue capsule nor are their borders sharply defined. The areas of calcification are nearly white in color and are small.

Plate No. II. Section of lung from Case No. 2. (One-half original size.) The lesions are usually surrounded by connective tissue and contain much calcification. There is a large cavity in about the centre of the tubercular area and numerous smaller ones are scattered throughout the lesion.

Plate No. III, Fig. 1. Portion of lung from Case No. 3.

(Two-thirds original size.) A little to the left of the centre of the specimen a tubercular mass may be noted. Other nodules are present around it and also along the lower and left hand borders of the specimen.

Fig. 2. Photomicrograph of tubercle from the lung of Case No. 1. (Magnified sixty diameters.) An area of calcification may be noted in the relatively large necrotic centre which is surrounded by the zone of lymphocytes in a connective tissue stroma. A capsule could not be made out.

PLATE No. I.

Section of Tubercular Lung From Case No. 1.

PLATE No. II.

Section of Tubercular Lung From Case No. 2.

PLATE No. III.

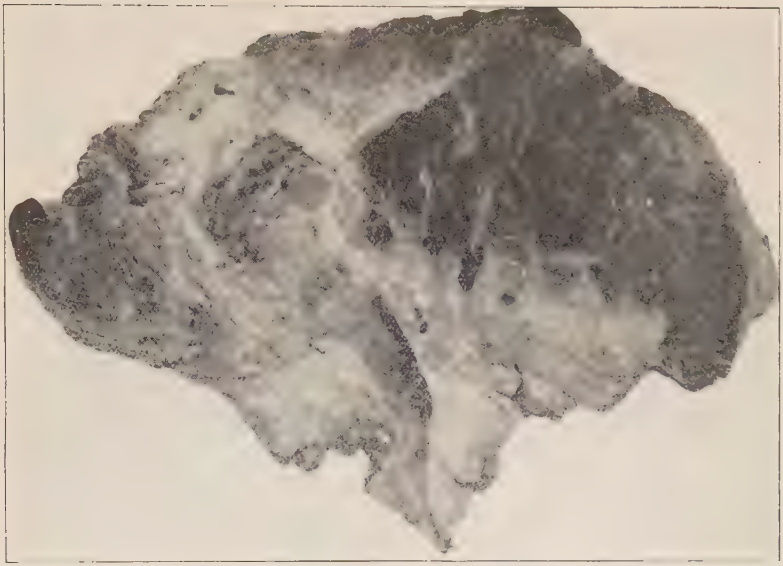

FIG. 1.— SECTION OF TUBERCULAR LUNG FROM CASE NO. 3.

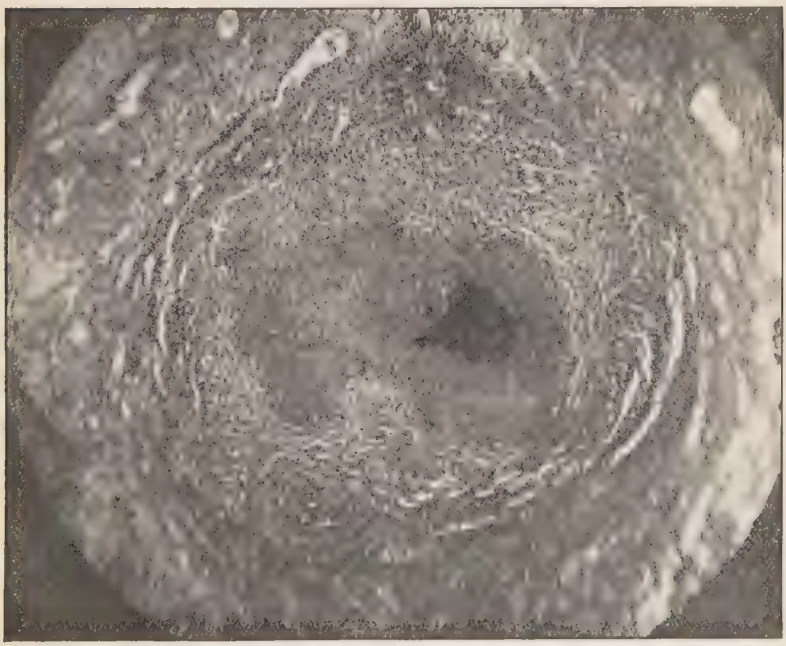

FIG. 2.— TUBERCLE FROM LUNG OF CASE NO. 1.

THE PATHOLOGY OF SPAVIN

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The term *spavin* apparently had its origin from the Latin *spavenius* used by Jordanus Ruffus in the middle of the thirteenth century. Originally it indicated various pathological processes in the neighborhood of the tarsal joint. Later the term became confined to disease processes occurring on the median side of the tarsus. Due to the different externally noticeable changes in the hock joint there are modifying terms used as prefixes to the term *spavin,* such as *bog, blood, moist, dry* or *bone, occult spavin,* etc.

There was at one time considerable confusion in the classification of spavin. DeSolleysel used the term *dry spavin* for lameness resembling spavin lameness but which did not originate from spavin. True spavin he named ox spavin. Saunier introduced the term *muddy spavin,* a form of spavin usually occurring in horses raised in damp, marshy localities. Gibson selected the term *bone spavin* because the swelling is of a hard consistency and also to distinguish it from a *soft blood spavin.* Bourgelat (1789) acknowledged only one form of spavin which he named *callous spavin.* By the term dry spavin he understood a form of lameness originating in the muscles and their nerves. He considered *ox spavin* as a swelling on the median side of the hock joint arising from stagnated lymph which is at first soft, later becoming hard as gypsum and very commonly occurring in oxen.

At the present time spavin is considered to begin by a rarefying osteitis in the subchondral bone, spreading to the joint to form erosions and ankylosis, and to the periosteum to form exostoses.

The knowledge of the pathology of spavin was, up to the beginning of the nineteenth century, very meagre. There were a large number of theories concerning the development of the thickening on the median surface of the hock joint. The basis of most of these theories is in harmony with the medical view, of humoral pathology, at that time.

Thomas de Gray in 1639 wrote, "A splint is, in the beginning, a very gristle. However, if it be long left alone, it will come to be

a hard bone or excression. A ring bone begins first with a slimy humor which in time groweth to a hard gristle. * * * A dry or bone spavin is a great hard crust as hard as a bone if it is let run, sticking or indeed growing to the bone."

Gibson (1754) places the origin of spavin in a soaking of the ligaments with moisture which condenses, forming a swelling composed of hardened glue which later grows like the callus of a fractured bone resembling a piece of flint without any visible pores, except the foramina for the passage of nerves and blood vessels such as are found in other bones that compose the skeleton.

According to von Sind (1770), strong muscular exertion produces an irritation in the vessels allowing the escape of the juices. These juices accumulate in the joint ligaments and on account of their corrosive nature act upon these parts producing a gradually hardening callus known as spavin.

Laffosse (1772) who places the cause and development of spavin in the same class as coronary ringbone, gives the changes as follows: "Following excessive work on heavily strained fibres, they lose their power of speed and cease to require the circulation of lymph. The latter stops, hardens, and causes a stiffness or tension which is at first of an inflammatory nature but soon becomes separated and a sort of horny growth results."

DeSolleysel (1775) thought that the bony swelling arises on the inner and lower part of the hock joint through a confluence of cold humours that gradually becomes bone hard by evaporation and dissolution of its delicate substances.

VonBusch (1788) wrote, "Spavin is indisputably no other than this lymph becoming thickened in many ways, and finally hardened. The ligaments, cartilage and tendons of the hind knee joint contain especially numerous circulatory vessels for the purpose of nourishment. The lymph contained in these vessels becomes tenacious by lengthy inactivity, sudden chilling, etc. It thickens and gradually accumulates. Thus, the spavin begins."

In 1794, Taplin wrote, "A splint is either an enlargement of the periosteum by an original rupture of small vessels, and the extravasated fluid collected and indurated by time or it is a callosity originally formed upon the bone and becoming ossified, constitutes a bony substance, seeming a deformed part of the bone

itself. * * * A bone spavin is exactly in a greater degree behind what a splint is acknowledged to be before."

Von Rohlwes (1801) treats spavin as follows: "Violent stretching of the binding tendons of the hock joint excites an inflammation at the points of attachment. An inflammation invariably causes an afflux of juices which bring about a thickening of the bone itself. Every bone is covered by a strong membrane. Should this membrane become injured, the bone becomes free to enlarge. Hence, whenever the membrane is injured by an inflammation brought about by an afflux of juices, the bone becomes thickened."

Havemann in 1805, was the first to show that in spavin the articular surfaces themselves are diseased. He says, "The cause of the lameness lies always in the diseased articular surfaces. The lameness begins as soon as the articular surfaces of the flat bones become denuded of cartilage. This is followed by a sort of growing together of the bones." He distinguishes between the hard or bone spavin, the soft or moist spavin, and the occult or invisible spavin.

VonArnim (1806) considers the cause of spavin similar to that of rheumatism, and a cure very difficult or impossible.

Von Hochstetter (1824) combines under the term spavin all harmful influences diminishing the strength or the flexibility of the hock joint. He considers forceful injury and swelling of the tibial flexor as the cause of bone spavin.

Dietrichs (1829) supports Havemann's view. He says that spavin is a local disease of the hock joint affecting the articular surfaces. He is of the opinion that the primary lesion is in one or more of the articular cartilages of the hock joint.

Regarding occult spavin he says, "The swelling is, in many cases, unimportant. It is hardly visible, and yet the animal shows marked lameness. This is usually the case when the spavin is observed in the process of development, at the stage when the articular cartilages mainly are affected and before the bones are grown together."

According to E. F. Gurlt (1831) spavin is a disease of the horse characterized by exostoses on the median side of the hock joint associated with ankylosis of the articular surfaces and ossifi-

cation of the ligaments. Like von Rohlwes, he considers that the bony outgrowth comes out of the bone itself following a disease of the periosteum. He says that too little is known about the changes in the cartilage to form an opinion. He asserts also that the growing together of the central with the third tarsus and the latter with the metatarsus usually occurs first.

Hering (1834) supports, in general, Havemann's ideas about spavin. He asserts, however, that not very seldom diseased articular cartilages are found which are not associated with lameness of the horse. Also that in *visible spavin* the articular surfaces are usually found unchanged.

Havemann's spavin theory is further supported by G. W. Schrader (1839). In his numerous investigations of diseased hock joints he found that, as a rule, the articular surfaces between the central and third tarsus are the first to suffer. This chondritis is followed by suppuration of the articular cartilages resulting in caries of the bones that are sooner or later united. According to his view the exostoses appear only after the articular surfaces are markedly diseased and the external border of the third tarsus has been affected by caries. He also points out the fact that many horses without exostoses show marked lameness, while others with exostoses show slight or no lameness.

Bartels (1843) claims that spavin originates with an inflammation of the tendinous attachment of the hock joint. In place of *visible* and *occult* spavin he uses the terms *tarsal* and *navicular spavin.* He used the former where the spavin is located in the upper part of the hock joint accompanied by long standing lameness; the latter, where the spavin is situated in the central and third tarsus not accompanied by lameness of the animal.

Bouley (1850) expressed his opinion that the affection of the articular surfaces is of marked importance in spavin. He says, "Spavin is not an external swelling situated at the articular margin of the flat tarsal bones alone. It is more than that. It is a constant expression of an internal lesion of the articulations of these bones as well."

According to Hertwig (1850) every bony outgrowth on the inner side of the hock joint should be considered as spavin. In very few cases this outgrowth begins as an acute inflammation of

the ligaments and the bone, brought about by external injuries to the hock joint. It begins more often as a chronic inflammation, principally of the bone. In such a hock joint the bone is found redder, more vascular, and more porous than normal. The periosteum is slightly thickened, and between the bone and periosteum there is found some coagulable liquid. Then the spavin outgrowth arises. In spavin without exostosis, the process begins in the articular surfaces or in the tissue of the bone itself. He holds that, besides strong exertions and kicks, rheumatism may be the cause of this affection. Later Hertwig said that the changes arising in the joint always follow a chronic arthritis.

According to Lawrence (1850) stiff joint or anchylosis arises from some accidental wound in the joint, in which case the synovia escapes outwards. This escape of the synovia occasions great irritation and inflammation, and if the orifice of the wound is not soon closed, the membranes begin to thicken, bony matter is thrown out from the heads of the bones, thereby uniting them in one mass, and the use of the joint is gone. * * * The inflammation from a wounded joint is very different from that which takes place in any muscular part. For as ligaments are not so vascular as muscles, they are consequently much slower in forming granulations, or, in other words, they possess not so perfectly the powers of regeneration.

E. Gurlt (1853) considers spavin a chonic arthritis affecting first the articulation between the central and third tarsus and later the ligaments that bind these bones and the periosteum. He remarks that the swelling and the ankylosing exostosis take place exclusively on the median side of the tarsus. The calcaneo-astragalar articulation as well as that between the astragalus and the central tarsus remain free even when they are surrounded by bony outgrowths.

O. F. W. Schrader, Jr. (1860) examined many hock joints of horses that he treated for spavin during life. He considers spavin to be a chronic arthritis with the following possibilities. The process may begin in the cartilage due to strong and continued injurious influences such as pressure which causes a weakening and destruction of the cartilage followed by an affection of the bones. The synovial membrane may be primarily diseased. This

causes a diminished amount of synovia or a diseased synovia which in turn affects the cartilage. Finally the subchondral bone may become primarily diseased by repeated jarring, pressure, etc. This causes a disturbed nutrition followed by necrosis of the cartilage.

Anacker (1864) claims that spavin is a chronic deforming and proliferating arthritis. He considers the changes in the cartilage as an amyloid process.

According to the researches of Stockfleth (1869) the inflammatory process in spavin may originate in the ligaments, or in the articular surfaces. In the former the symptom complex arises as a chronic inflammation of the capsular ligament. In the latter the seat of origin is in the median part of the articular surfaces whence it spreads slowly. The articular cartilage gradually disappears and the denuded ends of the bones may grow together.

According to Dieckerhoff (1875) spavin is a complicated chronic inflammatory process originating in the inner leaf of the bursa of the fanshaped muscular attachment of the tendon of the tibial flexor. From here it spreads to the capsular ligament and to the periosteum of the lower part of the hock joint producing a chronic synovitis, a softening and dissolution of the articular cartilage, and an inflammation of the bone marrow.

Among the etiological factors he mentioned a faulty histological setting in the structure of the hock joint as well as defective body structure, a peculiar temperament, and external influences, such as excessive exertions of the horse, especially excessive burdening of the posterior extremities.

In 1880, Gotti, after examining a large number of hock joints, mostly from horses showing spavin symptoms during life, concluded that spavin originates in the bones of the hock joint. He found that the process usually begins with a very slow osteitis of the central and third tarsus and of the metatarsus. In the course of this osteitis there is a widening of the Haversian vessels causing a nutritive disturbance of the bony connective tissue. The latter is finally transformed into plastic marrow tissue which produces a decalcification of the bone. The osteitis later leads to a chondritis characterized by a slow and continued inflammation of the articular cartilage accompanied by active proliferation and destruction of the ground substance.

In the inflammatory process of the bone, Gotti recognized two stages. A destructive period, during which the newly formed marrow tends to spread slowly, and a regenerative period during which the newly formed marrow tends to be transformed into compact connective or bony tissue. Should the proliferating process reach the articular surfaces, it is possible for the marrow elements to be transformed into osseous tissue and lead to a true ankylosis.

He considered the formation of osteophytes as secondary, usually following the disorder of the joint, and developing at the time when the changes have reached the margin of the articular surfaces.

According to von Klemm (1887) the etiology of spavin is as follows: "In all cases where the heel is trimmed low the hock joint is excessively extended through the forward pressure on the joint by the tendon of the flexor pedis. This is the case when the thigh is loaded. In connection with this extension there is undoubtedly an excessive tension of both tibial flexors which, with their five tendinous heads must, in their turn, have an injurious effect on the points of insertion around the hock joint. Should the horse be cow hocked or the median side wall trimmed low, the injury occurs only on the median tendons thus bringing about a spavin." He stated that in fifteen military horses whose heels were trimmed particularly low, nine showed spavin in from one to two months.

Bayer (1890) supported the views of Gotti.

Möller considers spavin as a *chronic deforming arthritis.*

According to Pflug (1892) the exostoses are the primary lesions of spavin. He considers them as arising from an inflammatory process, an *ossifying periarthritis* which is often complicated by an arthritis.

Hoffman (1892) at first supported Dieckerhoff's views of spavin. Later he declared that spavin is an inflammation of the bone, a hyperemia leading to a *rarefying osteitis* and finally to formation of lacunae.

Höhne considers spavin not as a primary disease of the hock joint but as a complication of gonitis.

Smith (1893) differentiates between an *articular* and a *non-*

articular form of spavin. He considers spavin to be a *chronic deforming arthritis.*

An interesting treatise on spavin appeared in 1893 by Aronsohn. He summarizes as follows:

1. Spavin begins with a periostitis brought about by an injury to the median terminal tendons of the tibialis anticus and by overstretching the long and short lateral ligaments. This periostitis leads to exostoses on the median surface of the hock joint.

2. The arthritis is mostly secondary and occurs only on both lower articulations of the hock joint.

3. The capsular ligament and the synovial membrane of this joint show only slight changes. An enlargement of the synovial villi is, macroscopically, not shown.

4. There is very little proliferation of the articular cartilage before degeneration takes place. Peripheral proliferation of the articular surfaces followed by ossification is never found. Eburnation of the denuded bone is likewise not seen.

5. Free bodies are never found in this joint.

6. Eventually, the disease process leads to a partial, rarely to a complete osseous ankylosis of the articular ends. The latter is never found in the tarso-metatarsal articulation.

7. Atrophy of the tarsal bones is rare. When it occurs, it is hidden by the articular ends and by the ossifying periostitis. The endosteal changes consist of an osteoporosis and an osteosclerosis. They do not influence the periosteal formation of bone.

8. The affection of the tibio-astragalar joint consisting of a fibrous thickening of the capsular ligament and a proliferating inflammation of the synovial villi is a secondary complication of spavin.

He admits that an arthritis may cause a periarthritis and all the above described changes; but he claims never to have noticed, in spavin, diseased articular surfaces without any changes in the periosteum. He holds that those cases where there are erosions on the edge of the articular surface without any periarthritic changes, these erosions indicate nutritive or functional disturbances without any important significance. He thinks it unlikely that the small joints of the tarsus, on account of their rigid structure and naturally limited movement, should be primarily diseased.

According to Eberlein (1898) the process begins by a reddening and softening of the subchondral bone of the central and the third tarsus, at the anterior and median part of the joint and 2 or 3 mm. from the margin. This spreads towards the articular cartilages to form erosions, and towards the periosteum to form exostoses.

The synovia is diminished to but a few drops. It is alkaline in reaction and of normal appearance. In cases of ankylosis, it thickens and gradually disappears. He did not find any bacteria in the synovia, in the bone, or in the cartilage.

The cartilage is at first changed in color. It is bluish red instead of whitish, and dull instead of shiny. Later there begins a superficial fibrillation followed by a deeper destruction of the cartilage. This becomes noticeable as punctiform erosions which later become confluent resulting in elongated erosions.

Microscopically the earliest change is an increased number of lymphoid marrow cells in the Haversian canals. These are at first lined along the walls of the canals. Later they are gradually increased in number until the canals are completely filled and they appear as proliferating granulation tissue. At the same time there is a disappearance of the bony tissue, indicated by a widening of the canals. This widening takes place in various directions so that the Haversian canals become irregular in shape. The dissolution of the bone is accomplished by numerous giant cells, osteoclasts, that originate, according to Kölliker, from the osteoblasts. This process is known as *rarefying osteitis* or *inflammatory osteoporosis.*

Soon after this resorptive process there appears a regenerative process in which there is a new formation of bone, known as *osteo-sclerosis* or condensing osteitis. This process tends to counterbalance the damage done by the previous process so that finally the bone becomes even denser than the original bone.

The osteo-porosis spreads to the neighboring parts and causes therein a destructive process. In this manner it reaches the articular surfaces. Here, however, it is checked by the articular cartilage. The granulation tissue then spreads out beneath the cartilaginous surface causing a disturbed nutrition of the articular cartilage. This is followed by a fibrillation of the upper and a softening of the deeper layers of the cartilage. Together, they

produce a splitting and a sinking of the articular cartilage. Usually, however, there occurs a true chondritis in the form of progressive and retrograde inflammatory processes. In this case the inflammation of the bone affects the cartilage directly producing a proliferation of the cartilage cells. The number of cells is increased and they lie together in groups. Between these groups of cells there are round or oblong, large multinucleated giant cells which produce a degeneration and resorption of the newly formed cells. This results in a loosening and fibrillation of the cartilage and in a destruction of the ground substance, and is eventually followed by a splitting of the cartilage. The proliferating granulation tissue very often simply penetrates the cartilage, i. e., it grows into the cartilage and dissolves it until it reaches the articular surface. Should the osteosclerosis now quickly follow, the chondritis may cease, and there may remain on the articular surface white punctiform elevations, the so-called *lime points*. In other cases the chondritis spreads rapidly and leads to a destruction of the articular cartilage.

There is often found ankylosis between individual bones, particularly between the central and the third tarsus. This growing together of the bones may be in the form of a pseudo-ankylosis produced by finger-shaped hyperostoses gripping one another, or it may be in the form of osseous ankylosis arising from the articular surfaces. The latter does not take place over the entire articular surface but in parts of it, so that on section of the joint there are seen elongated fissures in ankylosed parts of the joint. There is, therefore, only partial ankylosis as a rule and rarely only complete ankylosis. Eberlein claims never to have seen a fibrous or cartilaginous ankylosis, so that he regards ankylosis in spavin as being exclusively of an osseous nature.

Etiologically, he holds that spavin is due to pressure on the smaller bones of the hock joint. The extrinsic causes for this pressure are overstraining of the hock joint due to too quick movements, jumping, heavy loads, improper shoeing, etc. The intrinsic cause consists of an internal anlage due to faulty conformation of the hock, to temperament, and to the age of the animal. Heavy musculature in the pelvic region predisposes to spavin, particularly when the hock joint is not built correspondingly strong.

A long dorsum is also a predisposing factor. Horses with violent temperaments are more susceptible to spavin than phlegmatic horses. This is so because they attempt their work with greater exertion. Young horses are affected more often than older ones.

In affected joints the erosion is at first noticed 2 or 3 mm. from the margin. The reason for this lies, according to Eberlein, in the form of the articular surface. He claims that if pressure is exerted on a joint after it has been freed from its ligaments, it will be seen that the *closing border* is not at the margin of the joint, but a few mm. from it. On account of this fact, every mechanical lesion will exert itself more heavily on this closing border, so that the first inflammatory changes must occur on this part of the bone and cartilage.

This view that spavin begins in the subchondral bone is accepted to-day by most veterinarians. Hertwig was the first to point out this possibility and Gotti was the first to assert that it always begins in the subchondral bone. Williams claims that spavin is a local manifestation of a general disease.

Some French authors (Kitt) claim that diseases through which young animals pass predispose them to this condition.

Nathan's recent work on *arthritis deformans* is particularly interesting because it was supplemented by experimental arthritis in dogs. He injected a hemolytic streptococcus in the femoral veins of twelve dogs. Two of the animals died after 20 and 28 days respectively. The others were killed at intervals from the 3rd to the 90th day.

After a period of incubation of three days, practically all the animals showed more or less definite general symptoms. Only a few, however, had a marked rise of temperature which continued for three or four days, except in two cases that died.

In the dogs of this series the inoculation was followed by a surprising diversity of pathological changes in the epiphyseal ends of the bones. As the dose of the micro-organism injected was the same, this diversity was undoubtedly due to the variation in susceptibility, the stage when the animal was examined, the location of the epiphyseal infection, and the age of the animal.

In all cases the earliest epiphyseal change is either local or general hyperemia. There may be one or a number of foci. The ves-

sels are filled with blood, the veins are markedly dilated and later become thrombosed. At a time which corresponds to the period when the microorganisms escape from the blood vessels, the marrow cells in the neighborhood of the focus or throughout the epiphysis rapidly increase in number and soon more or less completely fill the marrow spaces. As the congestion and marrow cell hyperplasia increase, the bony trabeculae are gradually encroached upon and begin to undergo absorption.

The giant cells are markedly increased in number and are irregularly distributed in the medullary spaces. These cells are not located near the bony trabeculae, nor are there any large multinucleated cells which in any way resemble the cells described as osteoclasts to be seen in considerable numbers anywhere. These calls are the myeloblasts so often found in the granulation tissue, generally associated with osteitis. In this case at any rate the bone absorption is evidently not due to specific cells at all. The bone apparently disappears upon the approach of the newly formed vessels and the proliferating marrow cells. Here, as elsewhere, the bone makes way for encroaching soft tissue, and it is apparently the change in the vascular conditions or the resulting increase in the intramedullary pressure which is responsible for the bone absorption.

The focus may originate primarily within the marrow tissue or in the subperiosteal region of the epiphysis.

The earliest change in the vicinity of the epiphyseal focus is an intense hyperemia. Soon numerous hemorrhages appear rapidly followed by marrow and giant cell proliferation, and the marrow spaces in the involved area rapidly become filled with intensely engorged blood vessels and with lymphoid marrow containing numerous giant cells. As the process advances the bony trabeculae become absorbed and the adjacent marrow spaces become confluent.

The cell proliferation and bone absorption advance in all directions and encroach upon the epiphyseal line on one side and upon the joint cartilage on the other. When the inflammatory area encroaches but does not invade the chondral structures, there is some tendency to irregular cartilage cell proliferation. This stage of cartilage proliferation is, however, limited in extent and of

short duration. As the pathological process advances and actually encroaches upon the chondral structures, these, like the bony trabeculae, undergo absorption. Whether the cartilage undergoes local or complete absorption depends upon the virulence and the extent of the infection. With the absorption of the cartilage the intra-articular structures become involved in the morbid process.

The location of the central epiphyseal focus, and its tendency to extend, are subject to considerable variation. In the mild forms the process remains localized. Not infrequently the inflammatory area originates near the joint and extends toward the cartilage, finally penetrating this structure, within a more or less circumscribed area, without extensively involving the epiphysis. On the other hand there are cases in which the epiphyseal structure is extensively involved, though the process does not extend to the joint cartilage.

Very frequently the adjoining epiphysis also contained infective foci, or showed changes indicating that such foci had existed. In some cases there was, instead of a joint cleft, a continuous layer of granulation or connective tissue between the epiphyseal marrow of the two articulating bones without a vestige of cartilage between them. Occasionally there were ossifying areas in the connective tissue which bound the adjacent epiphyses together. In his opinion it is plausible that it is the gradual extension of ossification in the proliferating marrow which leads to bony ankylosis.

The subperiosteal epiphyseal focus begins as a localized inflammatory area in the region near the capsular insertion. This leads to local periostitis in a very short time. Frequently the disease advances from the periosteum to the capsule. Here hyperemia is succeeded by cell infiltration, finally by granulation and connective tissue formation leading to marked thickening of the capsule. The capsule may become adherent to the joint surface and subsequently the cartilage may become invaded by vascular loops and granulation tissue (the so-called panus). As a general rule, however, the inflammation invades the joint surface from the transitional area between the capsule, cartilage, and periosteum. When the condition is mild the cartilage resists the invasion of this panus so that in the final stage the joint surface is simply covered with non-vascular connective tissue. When, however, the condition is intense

or long continued, the cartilage becomes invaded and finally absorbed. This may be circumscribed producing a condition designated as ulceration. It may also be general and intense, the joint cleft completely obliterated and the opposing joint surfaces bound together by granulation tissue or in the final stage by connective tissue (fibrous ankylosis).

The joint cartilage plays a passive part in joint disease, irrespective of the origin or the course. It may be absorbed from above or from below, it may be completely replaced by granulation tissue, the absorption may be circumscribed causing a defect, isolated areas of apparently normal cartilage may remain within granulating or proliferating marrow tissue or more or less normal cartilage may exist under a layer of vascular connective tissue, but the cartilage itself never partakes of the inflammatory reaction. Like bone, it is passive and makes way for proliferating soft tissue.

These experiments were repeated with pneumococci and with staphylococci with practically the same results.

He concludes that all forms of arthritis are due to infection. He divides them according to the course into acute and chronic, according to the pathological lesions into active inflammations and proliferations, and into degenerations, and according to the location of the disease into osseous and subperiosteal or synovial forms of the disease.

He considers the joint effusions as symptomatic, and that the organisms enter the joint only after rupture of the cartilage. In early stages the organisms may be recovered from the epiphyseal bone marrow.

Since many authors consider spavin as an inflammation it would not be amiss here to briefly discuss inflammation. Inflammation, as defined by Burdon Sanderson, is the succession of changes occurring in living tissue when it is injured, provided that the injury is not of such a degree as to at once destroy its structure and vitality; or briefly, as *the local response to injury.*

An irritant may be sufficiently severe as to produce degeneration and death of tissue, but it may also be mild to such a degree as to stimulate the activity and produce a growth of the tissue. This is seen in an experiment of Ranvier. He injected weak solutions of silver nitrate into the peritoneal cavity of animals. At the end

of twenty-four hours the parts mostly affected showed an acute inflammation with complete loss of epithelium. In more sheltered parts, however, where the irritant had evidently arrived gradually and greatly diluted, the endothelial cells, instead of being lost, showed evidence of enormously increased activity and growth.

The structure of the synovial membrane is that of other serous membranes of the body. It is lined by endothelial cells forming a mesenchymal epithelium, and attached to the capsular ligament by areolar connective tissue, richly supplied with capillary blood vessels and lymphatics. So-called synovial villi are very commonly found in joints. However, the synovial membranes of healthy joints, more commonly seen in healthy young animals, are smooth and shiny and do not contain any synovial villi. Similar structures are very commonly found on the peritoneal surfaces, particularly in old animals. These are considered to be due to a mild bacterial or toxic irritation. It seems possible that the so-called synovial villi are also due to similar causes, and are not a part of the normal structure of the synovial membrane. Some authors, likewise, claim that the synovial membrane is not lined by mesothelium, at least not all over. It is possible that they were dealing with a desquamation of the mesothelial cells, since they found such cells in the synovia.

The nerves of the synovial membrane are described as terminating in peculiar corpuscles, *articular end-bulbs*. These are large, rounded or oval, flattened bulbs having a large core composed of small cells, and receive from one to four medullated nerve fibres terminating within them in fine convoluted and ramified non-medullated filaments. Nerves were traced into a plexus of pale fibrils lying close under the surface of the membrane. Pacinian corpuscles have also been noticed in many places under the synovial membrane.

The articular cartilage is of a hyaline type in which the cells and cell groups are smaller and more evenly dispersed, as a rule, than in other hyaline cartilage. These groups are flattened at and near the surface, and they lie parallel with it. Deeper and nearer the bone they are narrow and oblong, and are directed most vertically. Near the margin connective tissue is prolonged a certain way into the articular cartilage from the periosteum

and the synovial membrane and the cells acquire processes and present transitions to connective tissue cells, so that there is no sharp line of demarcation between the two tissues.

In the healthy state, no blood vessels penetrate the articular cartilages. Whatever nutrient fluid they require seems to be derived from the vessels of adjoining textures, especially the bone, and to be conveyed through the tissue by imbibition. Towards the circumference of the cartilage, however, underneath the synovial membrane, the synovial vessels form a narrow vascular border around it, named *circulus articuli vasculosus.* No nerves have been traced into any of the cartilages, and they are known to be destitute of sensibility.

In the non-vascular cartilage the inflammatory reaction would most likely resemble that of other non-vascular structures, such as the cornea. In this tissue a mild irritant produces destruction of cells followed by swelling and multiplication to make good the loss. More intense irritation causes a degeneration or death of cells, an accumulation of wandering cells, and a regeneration of the fixed tissue cells. Should the injury be near the periphery of the cornea, where there are vascular structures, there will be vascular changes in addition to the changes in the cornea.

In bone the only nerve endings found were those in the walls of blood vessels. Terminal blood vessels are described as being in the subchondral bone.

In classifying inflammations it seems advisable to speak of grades rather than varieties of inflammation since inflammation is a certain definite process. The appearance of the process varies with the nature of the tissue affected, the position of the tissues, and with the nature and the relative intensity of the irritant. Classifications of inflammation simply serve as a description of the changes that take place in given inflammations.

Of the acute inflammations of joints there have been described *serous, sero-hemorrhagic,* and *sero-fibrinous* synovitis and *suppurative arthritis.* The contents of the joint cavity, in acute cases of arthritis, are often turbid containing pus. These are *sero* or *fibrino-purulent* and *purulent synovitis.* In cases of short duration there are very often found reddened villous tufts on the synovial membrane composed of young connective tissue cells

and distended blood vessels and lined by mesothelial cells. These are cases of *subacute productive* or *proliferative synovitis.*

In addition to the synovitis there are often found, in acute cases, changes in the articular cartilage accompanied by hyperemia in the subchondral bone. These changes are manifested by changes in the staining reaction and diminution in the size of the nuclei. In other words, they are retrograde changes similar to those found in *degenerative* inflammations. In milder cases there may also be vascularization and disappearance of the articular cartilage, accompanied by vascular changes and by disappearance of the subchondral bone. In these cases the term *subacute erosive osteo-arthritis* seems to be justified.

Of the chronic inflammations of joints there have been described *serous* and *sero-fibrinous synovitis* and *chronic proliferative* or *productive synovitis* and *suppurative arthritis.*

In several instances, there were seen cases of chronic inflammations of joints in which there was a desquamation of the mesothelial cells lining the synovial membrane. These seem to justify the term *desquamative synovitis.* The following is a good example of desquamative synovitis.

Case 1. The right stifle of a six-year-old cow suffering from lameness for about two months. She was slaughtered on account of gonitis.

The joint was enlarged by an increased amount of a yellowish, slightly turbid synovia containing whitish flocculi. The capsular ligament was normal in thickness. The synovial membrane was covered in places by filiform whitish fibrous tufts varying in length from 1 mm. to 1 cm.

Microscopically these were composed of loose connective tissue containing numerous blood vessels. The connective tissue cells were mainly of the adult variety but there were also some fibroblasts. The blood vessels were of varying sizes. These tufts were covered by mesothelial cells. In some places these cells have been broken away from their basement membrane and are seen in detached clusters (fig. 1).

The articular cartilages of the femoro-patellar articulation were apparently normal.

In the femoro-tibial joint, the semilunar cartilaginous discs

were soft and friable. In places they have lost their cartilaginous appearance and resembled fibrous connective tissue instead.

Microscopically the nuclei were nearly all gone. The remaining protoplasm was arranged in the form of fibrils running parallel with the articular surface (fig. 2).

The articular cartilages on the tibial condyles were greatly thickened. The thickest parts were 5 mm. thick and were near the crest. They gradually diminished in thickness as they sloped away on each side of the crest. The affected areas were 2 cm. wide on each side of the crest and extended antero-posteriorly along the entire articular surface. In this part the cartilage was soft, roughened, and of a velvety appearance.

Microscopically the cartilage was composed of fibres running perpendicular to the articular surface. In the uppermost layer the nuclei have entirely disappeared. The cartilage cells were more nearly normal as they approached the subchondral bone. At the zone of provisional calcification the cartilage cells were nearly normal. This zone was intact. The subchondral bone showed slight hyperemia, otherwise it was apparently normal (fig. 3).

This is evidently a case of *chronic serous proliferative* and *desquamative synovitis,* and *fibrillary degeneration* of the cartilage.

In addition to the synovitis there are very often found changes in the articular cartilage and in the bones that make up the joint.

In the cartilage there may be a change in the staining reaction and a diminution in size of the nuclei. The cartilage may become thickened and fibrillated. In the early stages of fibrillation the cartilage is of the consistency of soft rubber gradually softening more and more until finally the cartilage disappears, producing an erosion. In the case described above the fibrillary degeneration of the cartilage is at a little earlier stage than that very ably described by Zalewsky. He claims that this condition originates in the joint while erosions not preceded by fibrillation of the articular cartilage begin in the subchondral bone. It is the erosive form of arthritis, according to Kitt, that leads to fibrous and osseous ankylosis.

Arthritis deformans is a peculiar inflammation of joints in man and often, perhaps erroneously, described as occurring in the

domestic animals. It is a chronic arthritis in which there is a series of changes in all the structures of the joint occurring at the same time. While there are in certain inflammations of joints of domestic animals, some changes that resemble those of *arthritis deformans*, that peculiar combination of changes has, as far as I have been able to determine, not been described in animals.

Technique.— The tibio-tarsal joints were opened and examined macroscopically. Sections were taken from diseased portions of the tibia and the capsule and fixed in Orth's fluid for microscopic study.

The intertarsal and tarso-metatarsal joints were sawed anteroposteriorly, through the middle, and the changes noted. The two halves were then sawed through laterally. This exposed to view the cross section of the periosteum and the capsular ligaments of the smaller joints of the hock.

The interosseous ligaments were now cut through and the individual bones separated.

Portions of the diseased bones 2 or 3 mm. thick, were placed in large quantities of Orth's fluid for five days changing the fluid as it became cloudy. They were then washed in running tap water for twenty-four hours, and placed in 67 per cent alcohol for the same length of time.

Decalcification was accomplished by means of 5 per cent concentrated nitric acid in 67 per cent. alcohol. Large quantities of the liquid were used and it was changed daily. This was continued until the bone became nearly transparent throughout.

The acidity was removed by means of running tap water, twelve hours.

The tissue was dehydrated by successive changes of 67 per cent., 82 per cent. and 95 per cent. alcohol. It was left in each for twenty-four hours.

It was then cleared in clove oil until the tissue was entirely transparent, about two or three days. An opaque area in the center indicates that the bone is not completely decalcified.

The tissue was then placed in the following solution for about a week: Celloidin or parloidin 1.5 gm.; oil of cloves 5 cc.; ether, 20 c.c.; absolute alcohol 1 c.c. It was imbedded in the same solution as follows: The tissue was placed in a vaseline lined

paper box and deeply covered with the solution. This was allowed to evaporate down in the air for about six hours adding celloidin as it became too low. It was then hardened under a bell jar in a dish containing some chloroform until it was sufficiently hard not to give way under finger impression. To obtain this consistency it was often necessary to allow the chloroform to act for about twenty-four hours, changing the chloroform once or twice in the meantime. The paper was now stripped off and the block trimmed to a little larger than the size of the tissue. It was then mounted on a wooden block 1 inch thick and slightly larger than the celloidin block. The surface of the wooden block was moistened with a few drops of ether-alcohol and immediately thereafter covered with the celloidin. The bottom of the celloidin block was now moistened with ether-alcohol and this surface put upon the prepared surface of the wooden block. This was allowed to stand in the air for about fifteen minutes and then hardened in chloroform as above. This block was placed in 82 per cent. alcohol ready for sectioning.

The sectioning was performed in the usual manner, the knife and the block being moistened with 82 per cent. alcohol. Sections prepared according to this method varied in thickness from 12 to 35 microns. The dimensions varied from about 1.5 cm. square to 6 cm. long by 4 cm. wide, a section including the two lower rows of tarsus, a portion of the metatarsus and the anterior and posterior ligamentous attachments of these bones.

The stains used were hematoxylin and eosin, and Mallory's connective tissue stain for special differentiation.

CASES

The discussion in this paper is based upon a study of sixty-five cases of spontaneous inflammations of joints leading to the changes described as spavin. In all the cases the gross appearance was carefully studied and microscopic sections were taken from various parts of the joints. On account of the necessity of shortening this article a number of the cases were eliminated. In these cases the changes were similar to those described here, so that they did not add materially to the discussion.

Case 1 was decribed on page 271.

Case 3. Hock joints from a pure bred percheron foal that died during delivery.

The right hock joint contained a small amount of yellowish synovia. The capsule was of normal thickness. The synovial membrane was covered in places by fine, reddened villous tufts. The edges of the articular surfaces of the tibia and of the astragalus were covered in places by a reticulum of blood vessels emanating from the synovial membrane, and extending into the joint for a distance of 4 mm. This growth was readily removed leaving the underlying cartilage smooth. The deeper parts of the articular cartilages were, however, vascularized in places. This vascularization extended from the subchondral bone

In the remaining articulations of the hock joint, the synovial membranes were covered by fine, reddened villous tufts. The interosseous ligaments were oedematous. There were reticular growths of vascular tissue covering the articular cartilages around the interosseous ligaments emanating from the surface of the interosseous ligaments and extending into the articulation for a distance of 3 mm. The thickest growth was in the articulation between the third tarsus and the metatarsus. All the cartilages were of a bluish color and free from erosions. All the bones of the hock joint were congested throughout.

The changes in the left hock joint were similar to those of the right hock joint and similarly located.

This is evidently a case of *subacute proliferative synovitis* and an *erosive osteo-arthritis* arising either from the bone alone or from both the bone and the synovial membrane.

The cause was probably an intra uterine hematogenic infection.

Case 4. A full grown foetus removed from a five year old cow that died of septicaemia, following traumatic gastritis.

The synovial membranes of nearly all the joints were reddened by hyperemia. In the left hock joint the synovia was reddened and the cartilages were of a bluish color. In the tibio-astragalar articulation the cartilages of both the tibia and the astragalus were covered at the periphery by reticular vascular growths emanating from the synovial membrane, and extending into the joint for a distance of 4 mm.

The right hock joint showed changes similar to those of the left, excepting that the synovia was yellowish.

The joint lesions in the cow were as follows: In the hock joints there were yellowish erosions in the tibiae and on the corresponding astragalar articular surfaces. In the stifle joints the synovia contained yellowish flocculi. There were several petechiae in the synovial membranes and whitish calcified points in the cartilages. The cartilage of the anterior and lower ends of the femurs were soft and roughened. There were no villous tufts on the synovial membranes.

In the foetus the case is apparently similar but not so far advanced as Case No. 3. In the cow there is apparently a *sero-fibrino-hemorrhagic synovitis* and a *degenerative arthritis* perhaps resulting from the *traumatic gastritis* or of an independent infection.

In the hock joints there is either a chronic erosive arthritis or a vestige of a previous arthritis.

Case 5. A bay foal that died of a pyemia with multiple abscesses of the kidneys and diffuse hemorrhage of the adrenals, at the age of thirty-six hours.

In all the joints the synovial membranes were reddened and the cartilages were of a bluish color. The synovia was of a brownish yellow color.

In the hock joints there were changes similar to those found in the other joints of the body. In addition there was a thickening of the cartilage in the centre of each tibial trochlear ridge. This thickening appeared white while the rest of the articular cartilage was bluish.

Microscopically the thickened area was composed of a non-vascular reddish material, probably fibrin, containing numerous fibroblasts with here and there an island of cartilage. On the surface it was covered by cuboidal mesothelial cells. In places it was separated from the articular cartilage. These places were lined by cells resembling flattened epithelium. The cartilaginous matrix of the upper layers of the cartilage were stained pinkish while that of the lower layers took a bluish stain. The cartilage contained numerous highly congested capillaries emanating from the subchondral bone (fig. 13).

In the subchondral bone, the marrow spaces were greatly enlarged at the expense of the bony trabeculae. They contained

numerous congested blood vessels surrounded by fibroblasts and some marrow cells. These consisted of lymphocytes, fat cells, polymorphonuclear leucocytes, eosinophiles, eosinophilic myelocytes, with here and there a giant cell.

Cultures taken from the joints and from other organs showed a growth of a short, thick, non-motile rod-shaped organism in pure culture. The colonies were greyish white, rounded, very viscid and the center was nucleated. It produced acid and no gas in dextrose, lactose and saccharose bouillon.

It did not coagulate milk or liquefy blood serum. There was no growth on gelatin at room temperature.

This is apparently a case of *acute erosive osteo-arthritis* of a hematogenic origin, caused by infection.

Case 6. A four-day Holstein heifer calf that died of a septicemia. Media inoculated from the joints and other organs gave a growth of B. coli in pure culture.

Both stifle joints, humero-radial joints, and hock joints were enlarged and distended by a dark colored synovia containing flocculi of fibrin. The synovial membranes of these joints were reddened by hyperemia.

This is a case of *acute sero-fibrinous synovitis* caused apparently by B. coli infection, either through the umbilical vein or intrauterine.

Case 7. Two black and white Holstein heifer calves received for diagnosis. One was about two weeks, the other about four weeks old. They showed symptoms of white scours and died from a septicemia of B. coli.

In the younger calf there was subcutaneous oedema and hemorrhage around both hock joints. In the anterior part of the astragalar trochlear grooves there were masses of reddened fibrin. Under the fibrin there was a large red erosion in each astragalus. Around this area the cartilage was becoming vascularized from the subchondral bone.

Microscopically in the most affected portion the cartilage was entirely absent. In place of it there was a layer of connective tissue with congested blood vessels coming from the subchondral marrow. This was covered by fibrin containing red corpuscles and a few leucocytes. Toward the periphery the cartilage was

partly gone and the remaining cartilage was covered by an exudate of fibrin and red cells. Further away the cartilage was being vascularized from the subchondral bone.

In the subchondral bone the blood vessels were increased in number and markedly congested. The marrow spaces were increased in size to such an extent that the bony trabeculae were entirely gone in places, and in other places were very much thinner than normal. The marrow spaces contained congested blood vessels, a large amount of fibrin, a few fibroblasts and a few marrow cells with only here and there a giant cell (fig. 6).

In the older calf the changes were identical with those of the younger one. In addition there was beginning erosions on the corresponding articular surfaces of the tibiae.

All the other joints of both calves were apparently normal.

These are cases of *acute fibrinous synovitis* and *acute erosive osteo-arthritis* caused by B. coli infection, probably through the umbilical vein.

Case 9. A week-old Belgian colt that died of a colon septicemia resulting apparently from navel infection.

The left hock joint contained a considerable amount of cloudy yellowish synovia containing flocculi of fibrin. The synovial membrane was reddened and thickened in places by a few red tufts. At the anterior end of the astragalus in the tibio-astragalar articulation, there was an area, near the attachment of the capsular ligament, that appeared as if the cartilage was being dissolved. The remaining articular cartilages were apparently normal.

The right hock joint showed changes similar to those of the left hock but not so far advanced.

Microscopically the synovia contained numerous polymorphonuclear leucocytes, a few lymphocytes, large mononuclear leucocytes, endothelial cells and rod-shaped organisms.

The stifle joints as well as the occipito-atloid articulation contained synovia similar to that of the hock joints. In addition there were petechiae on the synovial membrane in the right femoro-patellar articulation.

All the other joints were normal excepting that the synovial membranes were reddened.

This is a case of *acute fibrino-purulent synovitis* caused by B. coli infection that has entered through the navel.

Case 10. A week old bull calf died apparently of a toxemia or bacteremia resulting from pneumonia. Culture media inoculated from the liver, spleen, joints, and heart blood remained sterile.

There were reddened erosions in both hip, hock and carpal joints. All the other joints were apparently normal.

This is a case of *acute erosive osteo-arthritis* caused by infection.

Case 11. A heifer calf, two-weeks old, received for diagnosis. She died of acute hemorrhagic enteritis.

Both hock joints contained a reddish gelatinous substance. In the tibio-astragalar articulations there were reddened erosions 2.5 cm. long and 1 cm. wide on the tibial articular surfaces. The astragalar articular surfaces were normal.

Microscopically in the eroded area the cartilage was nearly gone. There remained a thin imperforated layer in which the matrix took a pinkish stain. There was some vascularization in the center as well as at the periphery of the erosion, by engorged capillaries emanating from the subchondral bone. In the cartilage that was not affected by erosion, the superficial half stained pinkish while the half toward the epiphyseal bone took a bluish stain.

In the subchondral bone the marrow spaces were enlarged and contained numerous engorged blood vessels, fat cells, fibroblasts, giant cells, various marrow cells and some fibrin. They were larger in the center of the epiphysis than they were towards the articular cartilage but the latter contained most of the congested blood vessels and the giant cells. The greatest amount of destruction of bone, however, was in the diaphysis just beneath the epiphyseal cartilage. Here there were a large amount of fibrin and a large number of endothelial cells in the marrow spaces in addition to structures found in the subchondral epiphyseal marrow spaces. Here also the marrow spaces coalesced due to absorption of some of the bony trabeculae.

Of the other joints of the body, in the hip joints, in the posterior metatarso-phalangeal, and in the elbow joints the articular cartilages were of a bluish color. In the stifle joints the synovial membranes were pinkish and contained hemorrhages, the cartilages were bluish and the contents were gelatinous. The remaining joints were normal.

This is a case of *acute erosive osteo-arthritis* resulting from scours in which the causative agent was apparently in the joint as well as in the subchondral bone.

Case 12. An about three-weeks-old Holstein bull calf that came for diagnosis with a history that calves were dying in convulsions. This animal died of a fibrino-purulent peritonitis apparently originating from umbilical infection. Culture media inoculated from the joints and other organs gave a growth of micrococcus albus and citreus.

In both hock joints the synovial membranes were covered in places by fine, reddened, villous tufts. There were large reddened erosions on the anterior part of the astragalar articular surfaces. There were two reddened erosions in the occipito-atloid articulations, one on each articular facet of the atlas. The remaining joints were normal.

This is a case of a *subacute proliferative synovitis* and *erosive osteo-arthritis* caused by a staphylococcus umbilical infection.

Case 13. A three-weeks-old heifer calf killed on account of scours. On autopsy there was found a fibrinous pleurisy and pneumonia as well as an acute catarrhal gastro-enteritis.

In the hock joints all the bones showed extensive erosions with the cartilages entirely gone in places. The joint cavities were filled with fibrin.

Both scapulo-humeral and both humero-radial joints were filled with fibrin and there was oedema around them. The humero-radial joints showed, in addition, small erosions in the humeral and the radial articular cartilages. The remaining joints were normal.

Cultures showed a micrococcus albus, a streptococcus and B. coli.

This is a case of an *acute erosive osteo-arthritis* and a *fibrinous synovitis* apparently caused by umbilical infection.

Case 14. A three-week-old cream colored foal that died of a purulent peritonitis as a result of umbilical infection.

Both hock and stifle joints were distended with a purulent exudate and the synovial membranes were reddened. The remaining joints of the hind limbs were normal.

The right scapulo-humeral joint and the carpal sheath were filled with a purulent and a fibrino-purulent exudate respectively. The left humero-radial joint and the synovial bursae of the perforans, perforatus and the extensor pedis tendon were distended with a fibrino-purulent exudate. The left carpal joint was distended with a purulent exudate.

The remaining joints of the fore limbs were apparently normal.

This is a case of an *acute purulent* and a *fibrino-purulent synovitis* apparently caused by umbilical infection.

Case 15. An about six-week-old calf that had been suffering from scours for some time and had been treated with serum. A week prior to death there developed an abscess on the left fore leg in the region of the joint between the first and second phalanx which was opened. On autopsy there was suppuration of the umbilicus, a fibrino-purulent pleuritis and an acute caseous and suppurative pneumonia.

Both hock joints contained reddish yellow flocculi in the synovia. There was a reddened erosion 2 cm. in diameter in each astragalar articular surface. The cartilages around these erosions were bluish.

The synovia of the right scapulo-humeral joint contained flocculi of fibrin. In the left humero-radial joint the cartilages were bluish, the synovial membrane was reddened and the synovia contained flocculi of fibrin. In the left pastern joint the ends of the bones were necrotic. The joint was open and contained fibrin and pus. In the occipito-atloid articulation, the atloid articular cartilage was of a bluish color. All the other joints were apparently normal.

Media inoculated from the joints and other organs gave a growth of a micrococcus and a gram-positive short motile rod. This organism did not produce gas in dextrose, lactose or saccharose. It did not liquefy serum or gelatin. Milk remained unchanged.

This is a case of an *acute sero-fibrinous* and *purulent synovitis*, and an *acute erosive osteo-arthritis*, caused apparently by umbilical infection.

Case 16. Left leg of a two-month old colt brought by the owner for diagnosis. The intestines contained numerous ascarids.

In the stifle joint the capsular ligament was thickened in places. On the median side of the femoro-tibial joint the capsule was completely ossified. The synovia was turbid and contained flocculi. The synovial membrane was almost completely covered by yellowish, filiform and flattened villous tufts varying in length from 1 mm. to 1 cm. In one place two parts of the synovial membrane were adherent by a smooth band of connective tissue 8 mm. wide, 2 mm. thick and 7 cm. long, running across the median end of the patella and attached to the lower border of the patellar articular cartilage (fig. 14).

In the femoro-patellar articulation, the femoral articular cartilage was thinner than normal nearly throughout. This area was studded with punctiform reddened erosions. The subchondral bone was reddened. The patellar articular surface was normal excepting a small erosion near the superior border and a fibrous attachment at the inferior border. Around this area the cartilage was covered by a thin reddened film of blood vessels. The external lateral ligament was macerated and appeared shreddy.

In the femoro-tibial joint the cartilage on the center of the median ridge was gone for an area of 3.5 cm. long and 2.5 cm. wide. The denuded bone was smooth and shiny and of a brownish gray color. The corresponding tibial articular surface was similarly affected for an area 2 cm. in diameter. The cartilaginous disc was gone at this point and at the periphery it was covered by villous tufts. On the lateral femoral trochlear ridge the articular cartilage was of a bluish color. There were punctiform erosions near the anterior and external border for an area 2 cm. long and 1 cm. wide. The corresponding tibial articular surface and that of the cartilaginous disc were normal.

In the hock joint the capsule was of normal thickness. The synovial membrane was covered nearly throughout by reddened fibrous tufts varying in length from 1 mm. to 1.5 cm. Most of these were filiform but some were flattened. Between the small bones, the synovial membrane was roughened by fine reddened tufts.

In the tibio-astragalar articulation there was an area 6 mm. square, in the centre of the tibial trochlear ridge, slightly roughened by very shallow, punctiform depressions. There was a similar area on the median side of this ridge.

Microscopically the cartilage contained areas where the nuclei were visible but did not take the nuclear stain. At the periphery of these areas there were nests containing numerous cells in which the nuclei stained deeply. These areas were near the subchondral bone.

The subchondral marrow spaces were enlarged and contained vessels engorged with blood and some fibroblasts.

The corresponding astragalar articular surface was more markedly affected. There was a purple shallow erosion 1.5 cm. long and 6 mm. wide in the trochlear groove. On the median side the erosion was slightly deeper, otherwise it was similar to the one on the corresponding tibial articulation.

Microscopically in the deepest part of the erosion the cartilage was thinner than normal. In other places there were necrotic areas in the cartilage. Some of these areas extended from the subchondral bone to the surface of the cartilage. Others were situated near the subchondral bone and did not extend to the articular surface. At the periphery of these necrotic areas there were nests containing numerous deeply staining cartilage cells.

In the subchondral bone the changes were similar to those of the tibial articulation but they were more marked.

The calcaneo-astragalar joint and the articulation between the astragalus and the central tarsus were bluish in places, otherwise apparently normal.

The remaining joints of this leg were normal.

Media inoculated from the joints gave a growth of B. coli.

This is a case of a *subacute proliferative sero-fibrinous synovitis* and *erosive osteo-arthritis* apparently caused by infection.

Case 17. A ten-week-old heifer calf killed on account of stiffness of the joints and general emaciation. On autopsy there were found fibrous adhesions in the pericardium and degeneration of the parenchymatous organs.

In the right hock joint, the capsule was thickened by a growth of fibrous tissue which was covered with fibrin. Microscopically this thickening was composed of granulation tissue on the outside next to the synovial membrane, and fibrin containing a few leucocytes on the inside, toward the joint cavity.

In the astragalar trochlear groove there was a grayish red

erosion 3 cm. long and 2 cm. wide. There were punctiform hemorrhages on the external trochlear ridge. The cartilage in that area was slightly roughened and lustreless. The corresponding tibial articular surface showed changes similar to those of the astragalus. The remaining articular surfaces of the hock joint were of a bluish red color, otherwise apparently normal.

Microscopically in the eroded areas, the articular cartilage showed different stages. At the periphery of the erosion a few cells, in several places next to the bone, were nearly normal. As they approached toward the articular surface, the cells gradually became smaller, the blue stain disappearing from the cell body. The nuclei also were becoming smaller, later being represented only by blue granules until finally, near the articular surface, the cells appeared homogeneous and of a pink color, with the nuclei entirely gone. In the eroded area the cartilage and the bone were replaced by newly formed capillaries, surrounded by fibroblasts with here and there an area of leucocytes. Further away, in the remaining bone, absorption has taken place. The marrow spaces were enlarged at the expense of the bony trabeculae. They were filled with leucocytes, giant cells, fibrin and a few fibroblasts. The vessels were distended with blood. At the periphery of the erosion the cartilage was vascularized.

The left hock joint was normal.

Of the other joints, the right coxo-femoral, the left stifle and the left carpal joints were affected similarly to the right hock joint. The right radio-carpal, carpo-metacarpal, metacarpo-phalangeal and the occipito-atloid articulations were similarly affected except that there were no erosions in the cartilages. The remaining joints were normal.

Media inoculated from the joints gave a growth of a pure culture of a streptococcus.

This is a case of a *subacute fibrinous synovitis* and *erosive osteo-arthritis* apparently caused by umbilical infection.

Case 18. An about ten-week-old Jersey heifer calf that suffered from scours. She died of an acute suppurative bronchopneumonia and an exudative peritonitis.

Media inoculated from the joints and other organs, except the lungs, remained sterile. That inoculated from the lungs gave a growth of streptococcus in pure culture.

Both hock joints were distended with synovia containing flocculi of fibrin. The synovial membranes were reddened by congestion. There was a reddened erosion 3 cm. long and 2 cm. wide on each tibial articulation. The remaining articular cartilages of the hock joints were bluish in color, otherwise apparently normal.

The other joints of the body were apparently normal except that the cartilages were bluish.

This is a case of an *acute sero-fibrinous synovitis* and *erosive osteo-arthritis* apparently due to infection.

Case 21. A five-month-old bay colt that died of gangrenous pneumonia, ten days after an operation on a large oedematous growth in the scrotal sac.

Both scapulo-humeral joints showed erosions 1 cm. in diameter on the humeral and on the corresponding scapular articular surfaces. In the humero-radial joints there were large triangular shaped erosions on the humeral and the radio-ulnar articular surfaces. In all the fetlock and the corono-pedal joints the synovial membranes were hemorrhagic and the articular cartilages showed small, rounded, reddened erosions. In the carpal joints the synovial membranes were hemorrhagic, otherwise the joints were normal.

In the right hock joint the capsular ligament was normal in thickness. The synovial membrane of the tibio-tarsal articulation was almost entirely covered with yellowish white villous tufts varying in length from 1 mm. to 1 cm. The synovial membrane of the intertarsal and tarso-metatarsal articulations was covered, in places, with short villous and flattened tufts. The interosseous ligaments were reddened by hyperemia.

There was a shallow bluish erosion 1.5 cm. long and 6 mm. at its widest point, in the center of the tibial trochlear ridge. There was a similar erosion 3 cm. long and 5 mm. at its widest point, on the corresponding astragalar articular surface.

In the calcaneo-astragalar articulation there was a bluish erosion 2 cm. long, 2 mm. wide and .5 mm. deep in the angle of the lateral astragalar articular facet. There was a similar but very shallow erosion on the corresponding calcanean articular facet. The remaining articular cartilages of the hock joint were of a bluish color otherwise apparently normal.

The left hock joint contained a little yellowish, turbid synovia containing flocculi of fibrin. The capsule was of normal thickness. The synovial membrane of the tibio-astragalar articulation was covered over half its surface by villous tufts varying in length from 1 mm. to 1 cm. In the center of the tibial trochlear ridge there was a slightly noticeable erosion 1.5 cm. long and 7 mm. at its widest point. The base of this erosion was covered by a thin layer of cartilage. On the corresponding astragalar articular surface there was a deeper bluish erosion 1.5 cm. long, 4 mm. wide and about 1 mm. deep.

In the calcaneo-astragalar articulation there was a purple colored erosion 1 cm. long, 2 mm. wide and .5 mm. deep in the angle of the lateral astragalar articular facet. The corresponding calcanean articular surface was similarly but very slightly affected. There was a flattened transparent growth of connective tissue projecting from the synovial membrane into this eroded area.

In the articulation between the astragalus and the central tarsus there was a flattened growth of connective tissue on the synovial membrane projecting into the joint cavity for a distance of 1 mm. There was a similar growth on the synovial membranes in the articulation between the central and the third tarsus and in the tarso-metatarsal articulation.

There was fibrous ankylosis between the central and the third tarsus that emanated from the space around the interosseous ligament and that ran antero-posteriorly for a distance of 8 mm. long and 2 mm. wide. The cartilage immediately surrounding the ankylosed area was depressed.

The remaining articular cartilages of the hock joint were bluish otherwise apparently normal.

Microscopically in the shallow erosions the cartilage was thinner than normal and covered by cells resembling flattened endothelial cells. The cartilage immediately underneath these cells resembled fibro-cartilage, and that toward the bone appeared normal. At the periphery of the erosion the superficial layer of cartilage took a pinkish stain while the deeper layers stained blue.

In the subchondral bone some of the marrow spaces were enlarged and filled with numerous very markedly congested blood

vessels, some lymphoid marrow cells and fat cells. Those further away contained few congested blood vessels and fat cells.

In the deeper erosions the microscopical changes were the same excepting more marked.

This is a case of *subacute proliferative synovitis* and *erosive osteo-arthritis* and an *acute hemorrhagic synovitis* and *erosive osteo-arthritis* in some of the joints. In the articulation between the left central and third tarsus there was a *chronic fibrous ankylosing osteo-arthritis.*

Case 22. A black pure bred percheron colt six months old killed on account of gonitis.

The left coxo-femoral articulation contained about a tablespoonful of clear yellow fluid. The cartilage of the cotyloid cavity was slightly roughened.

The left stifle contained 340 c.c. straw yellow, slightly viscid fluid with flocculi of a yellowish white color floating in it. The lateral trochlear ridge of the femur was eroded for an area of 8 cm. x 2.5 cm. At the rim of this erosion, near the attachment of the capsular ligament, the synovial membrane was gone for an area of 7 cm. x 4 cm. The ligament underneath was flabby and thready appearing as if it were chewed, due probably to its being caught between the patella and the trochlear ridge as the former slipped out of its place. The rest of the capsule appeared slightly thickened. The synovial membrane was thickened by fibrous tufts, the larger of which were about 5 mm. long. This was most pronounced at the insertion of the capsule and gradually disappeared away from that point.

At the upper edge of the erosion there were several pieces of cartilage of apparently normal structure and barely attached to it by what seemed to be threads of softened cartilage. The erosion presented a central area 4 x 1 cm. where the cartilage was entirely gone and the denuded bone was reddened and rough. Around this area the cartilage appeared as if it had been chewed off and was becoming smoothened probably by the friction of the slipping out of the patella.

The patella showed an erosion 2 cm. in diameter on the surface corresponding to the erosion on the lateral femoral trochlear ridge. On the synovial membrane surrounding the patella there were

fibrous tufts similar to those described above except for an area 5 cm. long at the external attachment of the capsule where the synovial membrane was normal.

In the right stifle there were subcutaneous hemorrhages, ecchymoses, for an area of 6 x 4 cm. and 100 c.c. of thick, turbid reddish brown fluid was removed from the joint. The capsule was divided into pockets so that some pockets contained clear straw yellow fluid while others contained the above mentioned cloudy fluid. The pockets were all tense and under pressure. The liquid was forced out, on incision, for a distance of about three feet. The cloudy fluid coagulated on standing.

The capsule was thickened by connective tissue. There was oedema around the capsule. The capsule contained ossified and chondrified areas, the largest one was 1 cm. x 5 mm. The synovial membrane was thickened by fibrous tufts some of which were threadlike while others were club-shaped or papilliform, most of them about 1.5 cm. long, projecting into the articular cavity. In the folds of the synovial membrane there were large flattened yellowish masses of coagulated fibrin that projected into the cavity. The edges of some of these were reddened. Some of these were free while others were attached to the capsule.

Microscopically, the masses lying free in the pockets of the synovial membrane were masses of granular, reticular and homogeneous fibrin. On the surface of this fibrin were quite a few leucocytes and endothelial cells. In this area were seen a few cocci and rods. The masses attached to the synovial membrane were composed of connective tissue cells surrounding small blood vessels. Here and there were seen minute areas of cartilage and of bone. This granulation tissue was covered by fibrin in which were seen some leucocytes.

The medial femoral trochlea was of a bluish color and the cartilage was partly covered by masses of fibrin, otherwise it was apparently normal. The lateral trochlear ridge presented an erosion at its lower half 4 cm. long, 2.5 cm. at its widest point and 1 mm. deep. In this area the cartilage was loosened for an area of 4.2 cm. It was of a yellow color, lustreless and studded with punctiform erosions.

Microscopic examination of the median trochlear ridge of the

femur showed that the articular cartilage was being dissolved in places by vascularization from the subchondral bone and by a marked fibrinous exudate. There were also a few giant cells and some endothelial cells. In the subchondral bone, the marrow spaces were enlarged to such an extent that few bony trabeculae remained. These spaces contained a marked fibrinous exudate and many congested capillaries. The marrow cells were more numerous nearer the articular cartilage than farther away.

The patella presented a small deep eroded area of 1.5 cm. x 8 mm. x 2 mm. on the surface which was applied to the erosion on the lateral trochlear ridge of the femur. On the same side and a little lower there was a mass of organized fibrin attached to the cartilage as if a line were drawn across it. This seemed to come from the synovial membrane.

Microscopically, the cartilage was apparently normal. This was covered by small blood vessels surrounded by connective tissue cells, i. e., granulation tissue. This granulation tissue varied in age, the oldest being next the articular cartilage and the youngest being covered by a thin film of fibrin about one-fourth the entire thickness of the band.

In the center of the erosion of the patella the cartilage was gone, the subchondral bone had been transformed into fibrous connective tissue cells surrounding congested blood vessels. On the surface of this tissue there was an exudate of fibrin containing bluish granules. The marrow spaces beneath the granulation tissue were enlarged and contained a considerable amount of fibrinous exudate, congested blood vessels, marrow cells and young connective tissue cells. Next to the bone they were lined by endothelial cells, a few giant cells and a considerable number of osteoblasts. The cartilage on both sides of the erosion was markedly degenerated, that on one side dipping into the granulation tissue. Blood vessels were seen penetrating the neighboring cartilage. There were a considerable number of giant cells subchondrally in this region.

In the femoro-tibial joint, the synovial membrane was thickened by reddened fibrous tufts. There were fibrinous masses in the folds resembling those in the femoro-patellar joint. There

was in addition a greenish yellow mass of fibrin resembling a lump of fat. The cartilaginous pads appeared normal.

In the right hock joint, the capsule was normal in thickness but the synovial membrane was thickened by filiform and a few papilliform fibrous tufts. In the tibio tarsal articulation there was a roughened erosion 1 cm. long, 5 mm. wide and 1 mm. deep on the median astragalar trochlear ridge. On section the bone beneath the erosion was reddened for an area of 2 mm. Anterior to this erosion the cartilage had been worn off for an area of 1 cm. long and 6 mm. wide. There were several punctiform erosions on the lateral trochlear ridge. There was also an erosion 3 cm. long, 4 mm. wide and 1.5 mm. deep in the trochlear groove.

Microscopically, in the center of the erosion on the median ridge, the cartilage was almost gone, but little connective tissue being in its place. On each side of this area the cartilage was replaced by connective tissue. There was also a replacement of bone by connective tissue. The fibrillae of the former were of a bluish tint while those of the latter were stained red. In the marrow spaces just beneath the articular cartilage there were proliferating capillaries distended with blood with here and there a giant cell. There was also some fibrinous exudate. In the area where the cartilage had been replaced by connective tissue, some of these vessels projected into this tissue. Near this area there were seen several nests of proliferating cartilage cells. The marrow spaces further away from the articular cartilage were markedly enlarged at the expense of the bony trabeculae. They were filled with fibroblasts, congested capillaries and some fibrinous exudate. (Fig. 5.)

In the calcaneo-astragalar articulation there was a shallow erosion 8 mm. long and 3 mm. wide in the angle of the lateral astragalar articular facet. There was a similar but smaller erosion on the corresponding calcanean articular facet.

In the articulation between the astragalus and the central tarsus, there was a flattened growth of connective tissue on the synovial membrane projecting into the joint cavity for a distance of 1 mm. There was gelatinous material around this growth.

The articulations between the central and third tarsus and between the third tarsus and metatarsus showed the same changes

as those in the articulation between the astragalus and the central tarsus. On section of the hock, the third tarsus was markedly reddened for an area of 3 cm. and throughout its thickness. There was a reddened area in the calcaneum forming a rim under its articular cartilage.

All the other joints of the body were apparently normal.

In cultures made from the joints a short streptococcus, staphylococcus albus and Ps. pyocyaneus, were obtained.

In this case, apparently, we have in the left stifle a marked *serous* and *proliferative synovitis* of long standing with a secondary *erosive osteo-arthritis.*

In the right stifle there was apparently an *acute sero-fibrinous synovitis* which was being transformed into a *chronic erosive osteo-arthritis* and *proliferative synovitis.*

In the right hock joint there was an *erosive osteo-arthritis* and a *proliferative synovitis.*

The cause seems to have been an infection perhaps through the navel.

The formation of bone in the fibrinous masses may be explained by the fact that lime salts are occasionally deposited in masses of old fibrin. These frequently exert some effect on the adjoining fibroblasts, in consequence of which these cells slowly invade and organize the lime salts, depositing them in their own intercellular substance and thus transforming themselves into true bone cells (Mallory).

In the bone the early process seemed to be an exudative osteitis.

Case 23. An eight month old black filly that died with a fibrinous pleuritis. The blood vessels were extensively infested with larvae of sclerostomes.

In the left scapulo-humeral joint there was a shallow, reddened erosion 7 mm. in diameter in the center of the humeral articular surface. In each humero-radial joint there was a reddened erosion 7 mm. in diameter on the inner border of the ulnar articulation. The synovial membranes were covered by fine reddish tufts. There were similar tufts on the synovial membranes of both fore fetlock joints. The cartilages of these joints were of a ish color. In the hind fetlock joints there were small reddish on each side of the ridges of the first phalanges. The

remaining cartilages were bluish. In the left hind pastern joint there was a small reddened erosion at the posterior edge of the second phalanx. In the right hind pastern the synovial membrane was covered by fine reddened tufts about .5 mm. long. The articular cartilages were bluish. In the carpal joints the articular cartilages were bluish.

In the right tibio-astragalar articulation the capsular ligament was normal in thickness. The synovial membrane was covered in places by yellowish villous and flattened growths varying in length from .5 mm. to 1 cm. In the center of the tibial trochlear ridge there was a bluish erosion 1 cm. long, 5 mm. wide and .5 mm. deep. In the corresponding astragalar groove there was a bluish erosion 4 mm. wide and 1 mm. at its deepest point running along the entire length of the groove. There was a roughened area 4 mm. long and 2 mm. wide on the lateral trochlear ridge.

In the calcaneo-astragalar articulation there was a bluish erosion 1 cm. long, 3 mm. wide and .5 mm. deep in the angle of the lateral astragalar articular facet. There was a similar erosion on the corresponding calcanean articular facet. There was a roughened bluish area 6 mm. long and 2 mm. wide on the median astragalar and calcanean articular facets.

In the articulation between the central and the third tarsus, the surfaces of both tarsal bones were roughened and bluish in color.

The remaining articular cartilages of the hock joint were of a bluish color, otherwise apparently normal.

In the left hock joint the changes were similar to those of the right hock joint and they were similarly located.

The remaining joints of the body were apparently normal.

Microscopically in the tibial erosions the cartilage was thinner than normal and the intercellular substance took the eosin stain. On the surface there were cells resembling endothelial cells. In the subchondral bone the marrow spaces were enlarged and contained markedly congested blood vessels, fibrin, endothelial cells, fibroblasts and fat cells. The cartilage outside this erosion was pink and the marrow spaces immediately underneath the cartilage were slightly enlarged and contained fibrin, endothelial cells, fibroblasts, some congestion and very few polynuclear leucocytes. In places the cartilage was just beginning to be dissolved by the

subchondral exudate. The marrow spaces away from the cartilage were filled with fat cells. In the articulation between the central and the third tarsus the synovial membrane contained numerous newly formed blood vessels, fibroblasts and several layers of mesothelial cells, some of which showed degeneration. In the roughened articular cartilage the intercellular substance took the eosin stain, the cell nests were large, and the cartilage was beginning to be dissolved in places by blood vessels from the subchondral marrow. In the subchondral bone, the marrow spaces were enlarged and filled with markedly congested blood vessels, endothelial cells, fibrin, fibroblasts, a few giant cells and fat cells. The bone next to the periosteum showed changes similar to those of the subchondral bone. In several places away from the capsular ligament, the periosteum was entirely gone and its place was taken by marrow similar to that of the subchondral bone. The subperiosteal enlarged marrow spaces contained more giant cells than the subchondral marrow spaces. The marrow spaces toward the center of the bones contained fat cells and slight hyperemia.

This is a case of *subacute erosive osteo-arthritis* and *proliferative synovitis* apparently due to an infection. The fibrinous exudate in the subchondral and the subperiosteal marrow spaces indicates an infection. While the greatest change here is in the subchondral and the subperiosteal bone, the general degeneration of the articular cartilage as well as the proliferation of the synovial membranes seem to indicate that there was irritation in the joint itself as well as in the bone.

Case 24. A yearling heifer calf that was killed on account of lameness in the left stifle joint.

In the left fore leg there was a yellowish roughened erosion 1 cm. long and 3 mm. wide on the scapular articular surface. In the humero-radial joint there was an erosion 1 cm. square on the radial articular surface. There were small erosions on the carpal articular surfaces. There was also an erosion on the median glenoid cavity of the first phalanx in the metacarpo phalangeal articulation.

In the left stifle joint there was an erosion 4 cm. long and 5 mm. wide extending across the trochlea on the distal end of the femur. The articular cartilage on the proximal end of the tibia was rough-

ened. There was an area 2 cm. in diameter where the cartilage was of the consistency of soft rubber.

In the left hock joint there was a long thread-like erosion extending across the tibial trochlea. In the calcaneo-astragalar articulation the calcanean articular cartilage was a little softer than normal. There was an erosion 4 cm. long and 1 cm. wide on the astragalar articular surface. The remaining articular cartilages of this hock joint were of a bluish color, otherwise apparently normal.

In the right hock joint there were three erosions, each 2 cm. long and 5 cm. wide, in the astragalar trochlear groove. There was also an erosion 5 mm. in diameter on the lateral trochlear ridge. The remaining articular cartilages of this hock joint were reddened otherwise apparently normal.

The remaining joints were apparently normal.

All the synovial membranes were normal.

Media inoculated from the joints gave a growth of micrococcus albus and citreus.

This is evidently a case of *chronic degenerative* and *erosive-arthritis* apparently caused by a micrococcus.

Case 25. The left hock joint of a two year old bay colt that died of inhalation pneumonia about ten days after an operation for cryptorchid.

The capsule was normal in thickness. The synovial membrane was thickened by reddened tufts varying in length from .5 to 8 mm.

The tibial trochlea showed a reddened erosion 2 cm. long, 5 mm. at its widest part, and about .5 mm. deep. The rest of the articular cartilage was bluish in color. The corresponding astragalar groove showed a reddened erosion throughout its length. It was 5 mm. wide and .5 mm. deep. The remaining astragalar articular cartilage was of a bluish color.

On section, the bone underneath the articular cartilage was markedly reddened, the reddest part being in the astragalus and calcaneum.

The calcaneo-astragalar articulation showed two small erosions on the external articular facet emanating from the space around the interosseous ligaments, and one erosion on the internal sur-

face apparently arising near the capsule and working inwardly. These erosions were on both of the corresponding surfaces of the astragalus and the oscalcis.

The articular cartilages of the articulation between the astragalus and central tarsus showed an irregular small erosion about 4 mm. in diameter.

The articulation between the central and third tarsus presented an erosion on the median border 1.5 cm. long, 4 mm. wide and varying in depth from .5 to 1.5 mm. the deepest part being on the central tarsus. Fibrous strands were present in these erosions. They were deepest at the extreme border, becoming shallower towards the center. The synovial membrane was thickened by fibrous tufts at this place. Near this place the synovial membrane was grown fast to the bone. The remaining articular cartilages in this part was roughened.

In the articulation between the fourth tarsal and the external metatarsal bones, the cartilages were eroded for a distance of 8 mm. in width running across the articulation from the capsule inwardly to the interosseous ligament. The remaining cartilages were of a bluish color.

In the tarsal sheath there was an erosion 1.5 cm. x 1 cm., 1 mm. deep on the posterior surface of the first tarsus near the median border. The base of the erosion was studded with punctiform hemorrhages. The remaining part of the tarsal sheath was normal.

Microscopically the eroded area in the tibial trochlea was covered by a very thin layer of cartilage composed of elongated nests of cells lying parallel with the articular surface. On the surface of this there were flattened cells, apparently fibroblasts and adult connective tissue cells. The subchondral marrow spaces, underneath the erosion, were enlarged and contained a few congested capillaries, fat cells and a few fibroblasts. The subchondral bone outside of the eroded areas was markedly denser than normal. There were practically no marrow spaces and the Haversian canals were small and contained slightly congested vessels. In the astragalus, aside from the eroded area, the cartilage and the subchondral bone were normal. In the erosion on the third tarsus the cartilage was entirely gone. In its place,

there was a thin layer of connective tissue. In the surrounding cartilage there were nests containing an unusually large number of cells. The subchondral marrow spaces in the eroded area were markedly congested. The remaining subchondral bone of the third and of the central tarsus was denser than normal.

This is a case of an *acute* or *subacute erosive osteo-arthritis* and *proliferative synovitis.* The dense condition of the bone may be a vestige of a previous condition, or the bones in this animal were naturally denser than usual. The fact that the density is uniform except under the erosions would tend to show that the bones in this animal were naturally denser than usual.

Case 26. A four year old Guernsey cow killed on account of lameness and general emaciation due to a chronic catarrhal enteritis.

In the right hind leg the coxo-femoral joint was normal. In the stifle joint there were erosions on the distal end of the femur on the facets that are applied to the semilunar cartilages. The erosions were shallow and extended about 2 cm. in diameter. The center of the semilunar cartilages showed fibrillation. The corresponding articular surface of the tibia showed fibrillary degeneration. There was a normal amount of yellowish viscid synovia in the joint. The synovial membrane was thickened by greyish tufts. The femoro-patellar joint was apparently normal, excepting a small erosion about 5 mm. in diameter on the lower border of the patella. Cultures taken from this joint remained sterile.

In the hock joint the cartilages of all the bones were eroded in places, the remaining catilage appeared reddened.

The other joints were apparently normal excepting that the cartilages were reddened.

In the left hind leg the coxo-femoral joint was apparently normal.

In the femoro-patellar joint the synovial membrane was roughened and covered by punctiform hemorrhages. Reddened fibrinous masses were present in the joint. These were adherent to the synovial membrane. The articular cartilages were of a bluish color and lustreless. There was a hemorrhagic area 2 cm. long and 4 mm. wide on the femur, above and immediately posterior to the articular cartilage.

In the femoro-tibial joint, the capsule was thickened. The synovial membrane was of a brownish color and it was thickened by papilliform tufts. The anterior surface of the external condyle of the femur was eroded for an area of 2 cm. by 1.5 cm. and about 2 mm. deep. This area was denuded of its articular cartilage. The denuded bone was smooth and shiny. Adjacent to and posterior to this area there was a similar eroded area from which the cartilage was not entirely worn away. On the internal side of the median condyle there was a shallow erosion about 1.5 cm. long by 5 mm. wide and about .5 mm. deep. The semilunar cartilages showed fibrillation in places. The tibial articular cartilages were roughened and of a bluish color.

Media inoculated from the femoro-patellar joint gave a growth of micrococcus albus, aureus and citreus.

The hock joint showed erosions in all the articulations.

The lower joints were apparently normal excepting that the cartilages were slightly reddened.

In the fore legs the elbow joints showed erosions about 2½ cm. long and 1 cm. wide on both the radio-ulnar and the humeral articulations. All the other joints were apparently normal.

This is a case of a *chronic erosive osteo-arthritis* and *proliferative synovitis.* There was also an *acute fibrino-hemorrhagic synovitis* of the left femoro-patellar articulation. The latter was evidently caused by a micrococcus infection. In the former the cause is unknown.

Case 27. A four-year-old brown mare that died apparently of a septicemia.

In both coxo-femoral joints the synovial membranes were thickened by reddened tufts.

In the right hock joint the tibio-astragalar articulation presented reddish and whitish tufts on the synovial membrane varying in length from 1 mm. to 1.5 cm. In places there were adhesions of different parts of the synovial membrane by these growths. In some places the synovial membrane was thickened by a reddened reticular growth. The tufts were filiform and papilliform. Some of the latter were of a yellow color containing fat. In the center of the tibial trochlear ridge there was a triangular-shaped, yellowish roughened erosion 1.5 cm. at its widest part

and 1 mm. deep. There was a similar erosion on the corresponding astragalar trochlear groove. On the anterior part of the median trochlear ridge of the astragalus there were two erosions each being about 1 cm. square and 1.5 mm. deep. The base of the anterior erosion contained a few strands of connective tissue. In the calcaneo-astragalar articulation there were several roughened and bluish areas on the articular cartilages, otherwise it was normal.

In the articulation between the astragalus and the central tarsus there were about twelve punctiform white calcified elevations on the median and anterior articular surface of the central tarsus. There were depressions on the corresponding astragalar articular surface. In the synovial membrane there was a flattened transparent growth, on the median side, projecting into the joint for a distance of 1 mm. There was a similar growth on the synovial membrane in the two lowermost articulations of this hock joint.

In the articulation between the third tarsus and metatarsus there was an erosion 4 mm. in diameter on the anterior border of the metatarsal articulation. In this erosion the subchondral bone was entirely denuded of the cartilage and remained smooth and yellowish. There was an elevation on the corresponding articular surface of the third tarsus. In this articulation a little towards the median side the capsular ligament was grown fast to the metatarsal bone for an area of 2 cm.

In the left hock joint the changes in the tibio-tarsal and in the tarso-metatarsal articulations were identical with those of the right hock joint. The remaining articulations were normal.

The remaining joints of the body were normal.

This is a case of *chronic proliferative synovitis* and *erosive osteo-arthritis*. The cause was not determined in this case.

This animal suffered from extensive arterio-sclerosis not produced by larvae of sclerostomes. This was probably due to micro-organisms, so that in this case there were probably bacteria present in the circulating blood which might have produced the changes in the joints.

The bones showed hyperemia throughout excepting for an area 3 mm. wide under the articular cartilages.

Case 30. An eight-year-old iron grey percheron mare that died with fistulous withers that extended to the spinal cord.

In both humero-radial joints there were erosions 2.5 cm. long and 2 cm. wide on the articular surfaces of the humera. There were similar erosions on the corresponding radio-ulnar articulations.

In the right hock there was a little yellowish clear synovia. The capsule was apparently normal. The synovial membrane was thickened by whitish villous tufts over about two-thirds of its surface. These varied from 1 mm. to 2 cm. in length. Some were filiform while others were flattened.

There was an irregularly-shaped bluish erosion 1.5 cm. long, 5 mm. wide and 1 mm. deep in the centre of the tibial trochlear ridge. On the corresponding astragalar trochlear groove there was an erosion 1 cm. square and 1 mm. deep on the anterior and lower border, and an erosion 5 mm. long, 2 mm. wide and .5 mm. deep near the posterior border. There was an erosion 1 cm. long, 6 mm. wide and 1 mm. deep on the median ridge of the astragalar trochlea. On the lateral ridge the cartilage was dull and softer than normal.

In the calcaneo-astragalar articulation, there was an erosion 1.5 cm. long, 1 cm. wide and .5 mm. deep on the external astragalar articular facet. There was a similar erosion on the corresponding calcanean articular facet. In the base of this erosion there was a whitish growth of connective tissue. There was an erosion 1 cm. long, 5 mm. wide and .5 mm. deep on the median astragalar facet and a similar erosion on the corresponding calcanean articular facet.

In the articulation between the astragalus and the central tarsus the cartilage was roughened along the anterior border varying in width from 3 mm. to 6 mm. Near the median border and immediately behind the roughened surface there was a thickened roughened area 1 cm. long, 5 mm. wide and .5 mm. at its thickest place. It was whitish in color. The corresponding articulation of the central tarsus was similarly affected. On drying the cartilage became dull yellow in color and extremely brittle, probably due to presence of lime salts in the cartilage. The astragalar and calcanean subchondral bone was reddened varying in width from 1 mm. to 5 mm., the reddening being 1 mm. away from the cartilage. The periosteum near the upper and anterior articular margin of

the central tarsus was thickened to about 1 mm. It was of a whitish color and soft in consistency.

The articular surfaces between the central and the third tarsus were roughened.

There was a fibrous ankylosis between the third tarsus and the metatarsus for an area of about 8 mm. long and 5 mm. wide at the anterior border of the articulation, not involving the capsular ligament. There was a groove about 1 mm. deep and .5 mm. wide around the fibrous tissue. There was an erosion 1 cm. long, 5 mm. wide and .5 mm. deep on the third tarsus at the anterior border of the interosseous ligament. There was a similar erosion on the corresponding metatarsal articular surface. The subchondral bone was reddened in places.

In the left hock joint the synovial membrane was not so extensively affected by fibrous tufts as that of the right hock joint. There was a similar erosion on the tibial trochlear ridge. On the corresponding astragalar articular surface the erosion in the trochlear groove was 3.5 cm. long, 1 cm. at its widest point and 1 mm. deep. There was an erosion 8 mm. in diameter and 1 mm. deep on the median astragalar trochlear ridge. There was extensive roughening of the cartilage around the latter erosion.

In the astragalo-calcanean articulation the changes were similar to those of the right hock and similarly located.

In the articulation between the astragalus and the central tarsus, the synovial membrane on the median side of the articulation was thickened by a flattened growth of connective tissue, projecting into the joint cavity for a distance of 2 mm. The articular cartilages in this place were roughened by shallow erosions for a distance of 2 cm. long and 1 cm. wide. The cartilage in this place was bluish. The remaining articular cartilage was apparently normal.

In the articulations between the central and the third tarsus and between the third tarsus and the metatarsus, the articular cartilages near the median border were roughened and bluish in color. The subchondral bones were reddened similarly to those in the right hock joint.

The remaining joints in the body were apparently normal.

Microscopically in the center of the right tibial erosion the cartilage was nearly all gone, but the lowermost layer remained.

Above that there were individual cartilage cells with a pinkish staining cell body and intercellular substance. This was covered by what appeared to be a mass of necrotic tissue in which the nuclei have disappeared. In this mass there were scattered fibroblasts. On the surface there was a thin layer of fibroblasts and adult connective tissue cells. The continuity of the lowermost zone of the cartilage has not been broken except in one point, at the periphery of the erosion, where it has been destroyed by subchondral congested blood vessels surrounded by fibroblasts. The cartilage in this place showed evidence of proliferation. There were masses of bluish staining cartilage cells and there were nests containing numerous cells. The cartilage above this area was degenerated but intact. In the subchondral bone under the eroded area the marrow spaces were enlarged and filled with fats cells, congested blood vessels and fibroblasts. Outside of this erosion the cartilage was nearly normal and the subchondral bone was somewhat denser than normal and slightly congested. A few of the subchondral Haversian canals were filled with congested blood vessels and fibroblasts.

In the left tibial erosion the cartilage was nearly all gone including the lowermost layer. There remained only a few cartilage cells in the center of the erosion. In the place of the cartilage there was a thin layer of fibroblasts and old connective tissue cells. At the periphery the bone projected nearly to the articular surface. The other changes were similar to those in the right tibia.

The right astragalar erosion was more advanced than that of the right tibia. It was deeper, involving the bone more, and the connective tissue layer was narrower. The subchondral bone and the surrounding bone and cartilage were similar to those of the tibia.

In the articulation between the central and the third tarsus there were foci of necrosis in the places that macroscopically appeared roughened. These foci involved in places the lowermost layers of the cartilage. In other places the foci of necrosis extended for a considerable distance into the subchondral bone. The cartilage covering these areas showed marked degeneration. The cartilage was broken only at one point where the bone was extensively involved. There was also degeneration of the car-

tilage near the synovial membrane. The subchondral bone aside from the necrotic areas was dense and the Haversian canals were, only here and there, enlarged and irregular in shape. All the Haversian canals were, however, filled with fibroblasts, adult connective tissue cells, and congested blood vessels. Some of the enlarged canals also contained fibrin and some lymphocytes and a few of them were filled with endothelial cells (fig. 7).

In the articulation between the central tarsus and the astragalus the cartilage next to the synovial membrane was thickened and showed fibrillary degeneration, that towards the center of the joint was thinner and degenerated. The subchondral bone was dense and congested. The neighboring synovial membrane was markedly congested and showed proliferation of mesothelial cells and blood vessels. The periosteum of the central tarsus was thickened by large cells, resembling cartilage cells, lying in rows parallel with the fibres of the attached ligaments. These cells were lying singly, and in very few places they appeared in nests of two, probably due to multiplication. In several areas the periosteum of the third tarsus was similarly affected, the other parts were normal.

This is a case of *degenerative, erosive* and *fibrous ankylosing osteo-arthritis* and *proliferative synovitis.* Some of the changes, such as fibrillary degeneration and the proliferation of the synovial membrane and of the periosteum, indicate a mild irritation. The other changes including the fibrinous exudate, and hyperemia and the necrosis in the subchondral bone indicate a severe irritation. They also indicate infection. It is possible that this condition was caused by metastasis of micro-organisms from the fistulous withers. This at first produced mild irritation and with the lowering of the resistance of the animal, the micro-organisms came to these joints in greater number and produced a severe irritation accompanied by the acute changes.

The degeneration without any break in the continuity of the artilages, and the proliferation of the synovial membranes seem to indicate that the irritant was perhaps first in the joint. The necrotic foci and the other changes in the subchondral bone seem to indicate that there were, perhaps later, metastatic foci in the subchondral bone.

Again, in the apparently unaffected parts of the articulations, the subchondral bones were denser than usual. This perhaps indicates that this density of the bone, the erosions in the tibio-tarsal articulations, and the tufts on the synovial membranes are vestiges of a previous inflammation. It may also be that in this animal the bones were naturally denser than usual. In the latter case the conclusion, that in this case the irritant was both in the subchondral bone and in the joint cavity or perhaps that it was in the joint cavity first and later in the subchondral bone also, may be justified. In the former case, where the assumption is that the mild changes are due to a previous inflammation, it is possible that in both inflammations the origin was in the subchondral bone.

Case 31.—A ten-year-old black gelding that died apparently of a streptococcic septicemia, after suffering with a sero-hemorrhagic colitis.

In both stifle joints the synovial membranes were covered by fine, reddened tufts. The articular cartilages were normal.

In the right hock joint the synovial membrane was thickened by reddened villous tufts, 3 mm. long. There was an erosion 3 cm. long and 2 cm. wide on the tibial trochlea. There was also an erosion 2 cm. long and 1 cm. wide on the corresponding astragalar trochlear groove. The remaining articular cartilages were apparently normal.

In the left hock joint the synovial membrane was thickened by reddened, villous tufts .5 mm. long. There was an erosion 2 cm. long and 1 cm. wide at the anterior border of the tibial trochlea. The remaining articular cartilages were apparently normal.

In the two hind fetlock joints the synovial membranes were thickened by reddened villous tufts 2 mm. long. There was a reddened erosion 3 mm. in diameter and .5 mm. deep in each joint on the posterior end of the groove on the articular surface of the first phalanx.

In both humero-radial joints there were a few tufts on the synovial membranes, otherwise they were normal.

In the fore fetlock joints the synovial membranes were thickened by fine reddened tufts. The articular cartilages were normal.

All the other joints of the body were normal.

Media inoculated from the hock joints gave a growth of a long-chained streptococcus and a micrococcus albus. Media inoculated from the heart gave a pure culture of a long-chained streptococcus.

This is a case of a *subacute proliferative synovitis* and *erosive osteo-arthritis* apparently caused by infection.

Case 33.— A twelve-year-old grey mare killed ten days after removing a large sized melano-sarcoma that involved the wall of the pharynx. On autopsy there was found extensive pneumonia in both lungs.

In both stifle joints the cartilages were of a bluish color and slightly roughened. The synovial membranes were normal.

In each humero-radial joint there was an erosion 2 cm. long, 1 cm. wide and 2 mm. deep in the groove of the humeral trochlea. There was a similar erosion on the corresponding radio-ulnar articulation. The remaining articular cartilage was of a bluish color.

In each tibio-carpal joint there was an erosion 1 cm. in diameter on the posterior end of the articular surface of the second carpus. The remaining articular surfaces were of a bluish color.

In the left hock the capsule was slightly thickened in places. The synovial membrane was covered in places by villous growths varying in length from 2 mm. to 1 cm. The longest tufts were at the points between the opposing bones.

There were two yellowish erosions, on the tibial trochlear ridge, one in the center, the other about 8 mm. posterior to it. The center one was 1 cm. long, 4 mm. wide, 1 mm. deep; the other one was 1 cm. long, 5 mm. wide, 1.5 mm. deep. There were several grooves on each side of the trochlear ridge running parallel with the joint. There were two erosions on the corresponding astragalar articular surface. The anterior erosion was 1.5 cm. long, 5 mm. wide and 1 mm. deep; the posterior one was 2 cm. long, 5 mm. wide and varying in depth from ½ to 1 mm. There were grooves on the astragalar articular surface similar to those on the corresponding tibial articular surface.

In the calcaneo-astragalar articulation, there was a yellowish erosion 1.5 cm. long, 3 mm. wide and .5 mm. deep in the angle of the lateral astragalar articular facet. This erosion emanated from the space around the interosseous ligament. There was a

similar erosion on the corresponding calcanean articular facet. The remaining articular cartilage of this articulation was bluish in places, otherwise apparently normal.

In the articulation between the astragalus and the central tarsus the articular cartilages were apparently normal. On the median surface of the synovial membrane, there was a flattened whitish growth of connective tissue on a line parallel with the opposing surfaces of the two bones, projecting into the joint cavity for a distance of 1 mm.

In the articulation between the central and the third tarsus, there was an erosion 3 cm. long, 2 mm. wide, .5 mm. deep running along the anterior and median border of the third tarsus. The base of this erosion was bluish white and slightly elevated. There was a similar erosion on the corresponding articular surface of the central tarsus. The synovial membrane in this articulation was apparently normal.

In the tarso-metatarsal articulation there was a reddened erosion 5 mm. long, 2 mm. wide and .5 mm. deep on the lateral and anterior border of the third tarsus, emanating from the space around the interosseous ligament. There was a similar erosion on the corresponding metatarsal articular surface. The cartilage around these erosions was bluish. The synovial membrane was not affected.

The remaining articulations of the hock joint were apparently normal.

In the right hock joint the capsular ligament was thicker than normal. There were whitish filiform and flattened tufts on the synovial membrane varying in length from 5 mm. to 1 cm. These completely covered the synovial membrane on the median side of the joint.

There were two yellowish erosions in the center of the tibial articulation. One of them was 1.5 cm. long, 1 cm. wide and 1 mm. deep. The other was 2 cm. long, 1.5 cm. wide and 1 mm. deep. There was a triangular erosion 2 cm. long and 1 cm. in its widest part at the anterior border of the astragalar trochlear groove. The articular cartilages were bluish and roughened. On section the subchondral bone was reddened.

The remaining articular cartilages were reddened, otherwise apparently normal.

The other joints of the body were apparently normal.

Microscopically in the eroded areas of the tibio-tarsal articulations the cartilage was entirely gone. The denuded bone was covered by a very thin layer of connective tissue. At the periphery of the erosions, the upper layer of the cartilage took a pinkish instead of a bluish stain. The subchondral bone as well as the bone beneath the eroded areas was denser than normal and congested. The marrow spaces, away from the joint, were filled with fat cells.

It is quite possible that the erosions and the fibrous growths in the joints of this case are remains of a former inflammation. The hyperemia in the subchondral bone is possibly due to metastasis following the operation.

Case 35. A fifteen-year-old red roan gelding that died with a gangrenous pneumonia.

In each scapulo-humeral joint there was a triangular erosion, 1.5 cm. at its base and sides on the head of the humerus. The remaining cartilages were bluish. In the humero-radial joints there were erosions 4 cm. long, 2 cm. wide and 3 mm. deep on the humeral articular surfaces. There were similar erosions on the corresponding radio-ulnar articular surfaces. The rest of the cartilage was bluish. In the carpal joints the synovia was reddish. The cartilages were bluish and eroded in places.

In the stifle joints the synovial membranes were thickened and roughened by a reticular growth of connective tissue.

On the outside in front and above the left hock, there was a swelling 3 cm. x. 2 cm. x 2 cm. composed of œdematous connective tissue.

The joint cavity contained about a tablespoonful of yellow viscid synovia. There was a small pocket on the external side containing a mass of partly organized fibrin.

The synovial membrane was thickened anteriorly by reddish villous tufts, about 1 cm. long. Posteriorly the synovial membrane was similarly affected except that the tufts were yellowish. The articular cartilages of the tibio astragalar joint were of a slightly bluish tinge in places. There was a very shallow erosion, 6 cm. long and 2 mm. wide, in the astragalar groove. There were two punctiform erosions on the corresponding tibial trochlear ridge. On section there was marked subchondral hyperemia in all the bones of this joint.

In the calcaneo-astragalar joint there was an erosion 2 cm. long, 1 cm. wide and 1 mm. deep on the lateral astragalar facet and a similar erosion on the corresponding calcanean articulating surface. This area was filled with a yellowish brown gelatinous mass. There was a smaller erosion on the median facets of these bones, partly filled with a similar mass. The remaining cartilages were bluish in places. There was a shallow erosion 6 mm. in diameter on the astragalus near the interosseous ligament and a similar erosion on the corresponding articular surface of the central tarsus. The articular cartilages of the third tarsus and of the corresponding articular surface of the metatarsus were roughened in places.

On the external surface of the right hock there was a fluctuating swelling 6 x 4 x 2 cm. on the median side posterior to the tibia just above the articulation. On opening, it showed that it was a dilatation of the articular capsule at that point. The contents were synovia and a free irregular mass 2.5 cm. long, 2 cm. wide and 3 mm. thick composed of shreds of fibrin mixed with synovia. There was also a partly organized mass of fibrin adherent to the synovial membrane. The synovial membrane was thickened by villous tufts that were anteriorly of a reddish color and posteriorly greyish. The dilated part also was lined by villous tufts. The articular cartilages of both the tibia and astragalus were of a slightly bluish color and contained grooves running from before backwards and parallel to each other. These grooves were 1 mm. to 6 mm. apart each being 1 mm. wide.

On section of the hock, there was marked hyperemia subchondrally in all the bones.

There were several shallow erosions in the calcaneo-astragalar articulation. The remaining cartilages were bluish in places, otherwise normal.

Microscopically in the erosion on the left astragalar trochlea the cartilage was thinner than in the other parts of the articulation. The remaining cartilage in the eroded area, as well as the surrounding cartilage, showed fibrillary degeneration. The subchondral bone was normal (fig. 9). In the right tibio-tarsal articulation, the grooves were of various depths varying from places that showed a dent in the articular cartilage to those in which the

cartilage has entirely disappeared leaving the subchondral bone denuded of cartilage. The remaining articular cartilage showed fibrillary degeneration. The cartilage showed in many places nests filled with numerous cartilage cells in which the nuclei were very small. The subchondral bone showed slight congestion.

In all the other joints of the body the cartilage was of a bluish color, otherwise apparently normal.

This is evidently a case of *chronic degenerative* and *erosive arthritis,* and *proliferative synovitis.* This originated apparently in the joints, since the subchondral bones are normal and there is no break in the continuity of the cartilage.

The bluish discoloration of the cartilages due to the subchondral hyperemia is probably due to metastasis from the pneumonia.

The erosions and the fibrous tufts are apparently vestiges of a previous inflammation.

Case 37.— An aged bay mare killed on account of extensive disease of the bones, ring bone, spavin, stiffness and possible ankylosis of vertebrae.

In the left hind leg, there was a flattened area 1 x 2 cm., on the articular surface of the coxo-femoral joint. Some of the articular cartilage was roughened and softened.

In the hock joint, the cartilages of the tibio-tarsal articulation were rough and eroded in places. About these areas there was some discoloration by blood. On section there was an area 2 cm. x 7 mm., including the internal half of the articulation between the third tarsus and metatarsus which was reddened, slightly porous and contained whitish streaks of connective tissue. The two bones were attached to each other at that point. On separation, the articular surfaces of the affected area were denuded of cartilage and slightly roughened.

Microscopically, the articular cavity was obliterated in several places. In these parts, the two approximating ends of the bones were grown together by connective tissue which contained at one point proliferating cartilage cells. In other places, the joint cavity contained some granular fibrin, the articular cartilages were degenerated and in a few areas the cartilage was replaced by connective tissue (fig. 17). In the subchondral bones, the Haversian canals were greatly enlarged and irregular in shape. They were

filled with fibro-blasts, some fibrin and congested blood vessels. The concentric lamellae were gone in most of them. At the periphery, next to the bone, they were lined by large deeply staining rhomboid cells (figs. 12 and 17).

The digital bursa was distended, elliptical in shape on the median and lateral side 2½ x 4 cm. The median distension was 5 cm. above the fetlock, the lateral was a little longer and bulged out more, 2½ cm. above the fetlock. The two were joined. On section a yellowish fluid escaped. Within the bursa there was œdematous fluid and fibrin. The wall was 2½ mm. thick. There were several hemorrhagic foci within the tendon sheath at this place. Microscopically, it was an *acute sero-fibrinous bursitis*, beginning to organize.

The marrow and upper half of the first phalanx was hyperemic. This extended to the metatarso-phalangeal joint. The cartilage was eroded in two places. There was some hyperemia in the marrow of the second phalanx. The joints seemed to be normal. The third phalanx was hyperemic in its lower third, reaching the anterior surface about the middle of the bone. The navicular bone seemed normal.

In the right hind leg, the articular cartilage of the coxo-femoral joint was softened and eroded in places. The femoro-patellar joint showed slight erosion of the proximal portions of the articular cartilage. In the femoro-tibial joint, the cartilage was roughened. There was exostosis on the anterior border of the calcaneum. The capsule of the tibio-tarsal joint was distended. There was a spavin with exostosis involving the median surface of the tarso-metatarsal joint. There was an exostosis on the lower end of the first phalanx, not interfering with the joint. The first phalanx was hyperemic beneath the metacarpo-phalangeal joint. The bone had a bluish tinge 2 mm. below the cartilage. The articular cartilage was eroded in two areas 3 mm. by 2 mm. situated in the central part of the joint. The lower two-thirds of the bone was apparently normal.

Microscopically, in the erosion most of the articular cartilage had disappeared. The remaining cartilage was of a homogeneous pinkish color with fibrous connective tissue cells scattered through it. The zone of provisional calcification was thinner at this place

and contained proliferating cartilage cells at the periphery of the erosion. In the subchondral bone, the Haversian canals were normal excepting very slight hyperemia. The articular cartilage around the erosion was fairly normal.

The second phalanx was apparently normal. The third phalanx was hyperemic for about half of its lower surface, the upper portion of the bone being apparently normal.

The left fore leg showed an enlargement of the perforans and perforatus tendons midway between the knee and fetlock. There was a productive inflammation with increase of connective tissue. The tendons were grown together for a distance of about 10 cm. The carpal joint was slightly enlarged on its anterior and inner side. The fluid in the carpal joint was of a brownish color. There were erosions on the distal end of the radius and on the corresponding carpal surface. The carpal check ligament was thickened and adherent to the adjacent tissue. The synovial membrane of the fetlock joint was discolored. The cartilage of the sesamoid bones showed erosion. There was also corresponding erosion on the articular surface of the metacarpus.

In the right fore leg, the fetlock joint had an erosion on the left side of the first phalanx, 3 x 5 mm. and about .5 mm. deep. There was an erosion on the right side of the sesamoid 1.5 x 5 mm. and .5 mm. deep, just through the cartilage. There was also an erosion on the metacarpus corresponding to that on the sesamoid. The lower end of the first phalanx was thickened and roughened by exostosis. On section, the marrow of the first phalanx was congested. This was especially pronounced at the upper third about 1 cm. beneath the articular cartilage. The second phalanx showed hyperemia in the medullary portion. In the medullary portion of the navicular bone, there was a reddened, softened area 2 mm. in diameter extending into the lower ventral part of the joint between the navicular bone and third phalanx. The medullary spaces were enlarged. Other surfaces of the navicular bone were apparently normal. In the third phalanx, the medullary portion was deeply reddened, extending to the surface of the joint and along the anterior border. The medullary spaces were enlarged.

All the other joints were apparently normal.

This is, evidently, a case of a *polyarticular chronic erosive osteoarthritis* with a *chronic fibrous ankylosing osteoarthritis* of both tarso-metatarsal joints. There was also in places a *chronic ossifying periostitis.*

Case 38.— An aged bay mare that died with swamp fever.

In the right hind leg, the articular cartilage of the acetabulum was of a yellowish color and roughened. The cartilage on the head of the femur was roughened. There was an erosion 1 x 2 cm. on the antero-internal side where the bone was denuded of its articular cartilage. On the opposite side of the articular head there were several slight depressions in the cartilage 2 mm. in diameter. In the center of these depressions there were small yellowish white calcified areas 1 mm. in diameter. The cartilage on the lateral femoral trochlear ridge was eroded at the ligamentous attachment. Otherwise the stifle joint was apparently normal. In the hock joint there was an erosion 1.5 x 1.2 cm. and 1 mm. deep on the tibial trochlea and a corresponding erosion on the astragalar trochlea. There was also an erosion 1 cm. x 5 mm. and about .5 mm. deep on the calcaneum and a similar erosion on the corresponding astragalar articular surface. In this erosion there was a gelatinous mass, apparently the remains of the cartilage. The cartilage surrounding this erosion and the remaining intertarsal cartilages were of a bluish color, otherwise apparently normal. On section, the subchondral bone was slightly reddened. There was a partial osseous ankylosis between the middle and lower rows of tarsus, as well as between the lower row of tarsus and the metatarsus. In these places the articular cartilage was gone and the entire mass of bones were inseparably fused together so that the joint appeared as one bone, excepting for two areas each 1 cm. long in the intertarsal and one area 8 mm. long in the tarso-metatarsal articulation, where the articular cartilages were still present. (Fig. 11.) Anteriorly there was a spindle shaped area 2 cm. long and 5 mm. at its thickest point projecting over the tarso-metatarsal articulation. It was composed of rather porous bony substance. It was apparently an *ossifying periarthritis.*

Microscopically, what remained of the articular cavity was filled with a sort of a hyaline substance taking a greenish yellow stain. The articular cartilages appeared homogeneous, without

any nuclei and of a pink color. The zone of provisional calcification was represented by a homogeneous blue line. In the subchondral bone the Haversian canals were enlarged, of an irregular shape and were filled with adipose tissue and congested blood vessels. Those further away contained fat globules only. In the cortical bone, in the area where the articulation should be, many of the Haversian canals were of a normal appearance. Some of the canals were enlarged, of an irregular shape and were filled with congested blood vessels and adipose tissue. (Fig. 18.) The periosteum and ligaments were apparently normal.

Of the joints on the left side of the body, the hock was the only one examined. Here the changes were similar to those of the right hock joint and they were similarly located.

In the right foreleg there was an erosion 2.5 x 1 cm. and 2 mm. deep on the humeral articular surface. There was a similar erosion on the corresponding radio-ulnar articular surface.

All the other joints of the right limbs were apparently normal.

This is a case of a *chronic erosive polyarthritis* and a *chronic osseous ankylosing intertarsal* and *tarso-metatarsal osteoarthritis.*

Case 39. An aged sorrel mare that died with a ruptured diaphragm.

In the left scapulo-humeral joint there was a yellowish erosion 1 cm. in diameter and 1 mm. deep on the scapular articular surface. There was a similar erosion on the corresponding humeral articular surface. In the humero-radial joint there was a large yellowish erosion on the radio-ulnar articular surface and a similar erosion on the corresponding humeral articulation. On the latter articular surface there were also small scattered erosions varying in size from 2 mm. to 1 cm. in diameter and .5 to 2 mm. deep. In the fetlock joint there were small depressions 1 mm. in diameter in the groove of the first phalanx. In the center of these depressions there were whitish calcified points. The remaining articular cartilage was apparently normal.

In the right hock joint the synovial membrane was thickened by villous tufts. There was a yellowish erosion 2 cm. long, 1 cm. wide and 1 mm. deep in the center of the tibial trochlear ridge. There was a yellowish erosion 4 cm. long, 3 mm. wide and .5 mm. deep, in the astragalar trochlear groove. There was an erosion

1.5 cm. x 1 cm. and .5 cm. deep on the internal face of the lateral trochlear ridge. Surrounding this, there were several punctiform erosions and one white calcified point about .7 mm. in diameter.

In the calcaneo-astragalar articulation there was a whitish and yellowish erosion 2 cm. long, 5 mm. wide and .5 mm. deep on the lateral astragalar articular surface. There was a yellowish erosion 5 mm. square and .5 mm. deep on the median astragalar articular surface. There were similar erosions on the corresponding calcanean articular surfaces. The remaining articular surfaces were bluish in places, otherwise apparently normal.

Microscopically, in the erosions the zone of cartilage next to the bone was the only cartilage left. The rest of the cartilage was gone. Its place was taken by a homogeneous pinkish staining substance containing connective tissue cells. At the periphery of the erosion the upper layers of cartilage cells were degenerated. In the subchondral bone the marrow spaces were enlarged and filled with fat cells and congested blood vessels.

In the left tibio-astragalar articulation the synovial membrane and the tibial trochlea showed changes similar to those in the right hock joint. There was a yellowish erosion 3 cm. long, 1.2 cm. at its widest point and 1 mm. deep in the astragalar trochlear groove. There was an erosion 6 mm. in diameter in the middle of the median trochlear ridge. The subchondral bone of this articulation was reddened.

In the calcaneo-astragalar articulation there was an erosion 8 mm. long, 5 mm. at its widest point and .5 mm. deep on the lateral calcanean articular facet and a similar erosion on the corresponding astragalar articular surface. The median articular facets were normal except at the lower border for an area of 1 cm. long and 5 mm. wide where the cartilage was of a purple color.

The articulation between the astragalus and the central tarsus was normal excepting a few spots where the cartilage was of a purple color. These spots were irregularly distributed over the entire articular surface.

In the articulation between the central and the third tarsus, the anterior and median margins of the articular cartilages were eroded. The borders of the erosions were irregular. In some places there were depressions and in others there were elevations.

The elevated areas in the erosions were due to a growth of whitish connective tissue. In places the two bones were grown together by connective tissue. In these places the synovial membrane was grown fast to the bones. This area was about 8 mm. wide at its widest point.

In the center of the third tarsus there was a yellowish, roughened, raised calcified area 1 mm. in diameter. There was a depression on the corresponding articular surface of the central tarsus. There was a shallow, roughened erosion 5 mm. in diameter in the posterior part of the third tarsus and a similar erosion on the corresponding articular surface of the central tarsus.

In the articulation between the third tarsus and the metatarsus the anterior border was roughened for a distance of about 2 mm. in width. Otherwise the articular cartilages were normal.

The hind fetlock joints were normal except that the synovial membrane was covered with reddened villous tufts. In the left hind leg in the articulation between the first and second phalanges the two bones were grown together by partial *osseous ankylosis.* There was also a *peri-articular paraostosis* in the form of a smooth bony growth 1.5 cm. thick and 5 cm. long. Between the bones and the bony outgrowth there was a thin layer of connective tissue. There was a ringbone in the right hind leg on the distal end of the first phalanx not involving the articulation.

The right fore leg was not examined.

The remaining joints were apparently normal.

This is a case of a *chronic erosive osteo-arthritis* and *proliferative synovitis* with a *chronic fibrous ankylosing osteo-arthritis* of the left hock joint and a *chronic osseous ankylosing osteo-arthritis* with a *chronic ossifying peri-arthritis* of the left phalangeal joint. The subchondral and subsynovial hyperemia was probably due to the cause that produced the death of the animal.

Case 40. An aged bay mare that was killed on account of chronic pulmonary emphysema.

In both coxo-femoral joints the articular cartilages of both the cotyloid cavities and the corresponding heads of the femurs were thickened, wrinkled, and of the consistency of soft rubber. The synovial membranes were normal.

In the left hock joint the synovia was normal. The capsular ligament was normal in thickness. The synovial membrane was thickened and roughened by a reticular growth of connective tissue.

In the center of the tibial trochlear ridge there was an erosion 1 cm. long, 5 mm. wide and .5 mm. deep. In the base of this erosion there was a growth of whitish connective tissue. There was a similar erosion in the corresponding astragalar groove. There was considerable roughening of the cartilage on the lateral articular surfaces of this joint.

In the calcaneo-astragalar articulation there was an erosion 1 cm. long, 4 mm. wide and 1 mm. deep in the angle of the lateral astragalar articular facet. The base of this erosion was yellowish containing reddish points in several places. There was a similar erosion on the corresponding calcanean articular facet. This erosion emanated from the space surrounding the interosseous ligament.

The articulation between the astragalus and the central tarsus was normal.

In the articulation between the central and the third tarsus there was slight roughening on the anterior and median margin of the articular cartilages. There was fibrous ankylosis near the posterior end of the interosseous ligament for an area of 4 mm. in diameter. The synovial membrane of this articulation was normal.

There was fibrous ankylosis between the central tarsus and the os tarsale I et II at the upper border, for a distance of 2 mm. in diameter involving the synovial membrane. The cartilages around the ankylosed area were roughened by erosion. The remaining articular cartilages of the hock joint were normal.

In the right hock joint the tibio-astragalar articulation was similar to that of the left, except that there was not so much connective tissue in the erosions. There were also a few reddened fibrous tufts on the synovial membrane in addition to the thickening of the latter by reticular connective tissue.

The calcaneo-astragalar articulation was similar to that of the left hock except that the erosion contained a growth of whitish connective tissue.

In the articulation between the central and the third tarsus there was fibrous ankylosis near the posterior end of the interosseous ligament for a distance of 5 mm. long and 2 mm. wide running in an antero-posterior direction.

There was fibrous ankylosis between the central tarsus and the os tarsale I et II similar to that of the left hock joint.

The remaining articular cartilages of the hock joint were normal.

The remaining joints of the hind limbs were normal. The fore limbs were not examined.

Microscopically in the ankylosed area the synovial membrane was obliterated. The bones were grown together by a homogeneous non-vascular mass containing degenerated cartilage cells and connective tissue cells. Some of each subchondral bone has been replaced by that tissue. There was, however, a line of demarcation between the bone and this mass of tissue. This line stained blue and was continuous on both sides of this mass except at one point where it was broken by an enlarged Haversian canal. This blue line was composed of cartilage cells which in places showed evidence of proliferation. In the part of the point that was not grown together the upper layers of the articular cartilages, on both surfaces, were necrotic. The layers next to the subchondral bones were degenerated. In the subchondral bones the Haversian canals were enlarged and irregular in shape. They were filled with fibrin, congested blood vessels, newly formed blood vessels, endothelial cells, fat cells, and fibroblasts. The periosteum in the vicinity of the ankylosed area was thickened by large cells lying in rows parallel with the fibres of the attached ligaments.

This is a case of a *chronic erosive* and *fibrous ankylosing osteoarthritis,* and *proliferative synovitis.*

Case 41. The hock joints of an aged horse that died of swamp fever.

In the right hock joint, there was a bony outgrowth, 3 cm. long, 2 cm. wide and 1 cm. thick, on the median side over the articulation between the upper and middle rows of tarsus. The articulation between the astragalus and calcaneum was obliterated throughout its extent excepting for a small area 5 mm. long at the upper border. The obliterated part was reddened throughout

its extent, the articular cartilages were apparently gone and in places it seemed as if the two bones were grown together by fibrous tissue. (Fig. 10.) In the articulation between the astragalus and the central tarsus there was an area 1 cm. long immediately anterior to the interosseous ligament where the articular cartilages were gone and the bones were grown together by fibrous tissue. There was slight hyperemia around this area. The subchondral bone was reddened for an area extending from this ligament to the bony outgrowth on the median side. On the other side of the ligament, the subchondral bone as well as the articular cartilages were apparently normal. There was a very shallow, uneven erosion 12 mm. long and 5 mm. wide in the astragalar trochlear groove in the center of which there were whitish areas of connective tissue. There was no reddening of the subchondral bone in this eroded area.

On the tibial trochlear ridge there was a whitish area, 3 cm. long, 6 mm. wide and 3 mm. deep that appeared as if an erosion was in this place and it had become healed so that now the ridge was smooth throughout its extent. In the median trochlear groove there was a white growth of connective tissue on the articular cartilage 15 mm. long and 5 mm. wide. There was a growth 3 cm. thick on the median side of the tibia immediately above the articulation. It was composed of fibrous tissue and cartilage.

Microscopically in the periarticular bony outgrowth the marrow spaces, in the central part of the newly formed bone, were large and filled with congested blood vessels, fibrin, fibroblasts and lined with fibroblasts and here and there a giant cell. In place of the synovial membrane there was an area of cartilage cells arranged in rows perpendicular to the articular surfaces. Next to this area the two articular surfaces were grown together by cartilage and connective tissue cells. Toward the center of the articulation there was a considerable amount of pinkish staining homogeneous substance apparently the remains of the upper layers of the cartilage. The cartilage next to the subchondral bone was intact except near the edge of the articulation. The periosteum on each bone under the newly formed bone was intact. In the subchondral bone at the articular border, the Haversian canals were enlarged and filled with fibroblasts and congested blood

vessels. Toward the center of the articulation the Haversian canals were enlarged and filled with congested blood vessels and fat cells. On the outside the newly formed bone was covered by a large amount of connective tissue.

In the calcaneo-astragalar articulation, at the proximal border the upper layers of the cartilage were pinkish and homogeneous and the two articular cartilages were separated by the joint space. Approaching toward the center of the articulation the cartilages were the same but the joint space was filled with connective tissue cells and fibroblasts. Further down the necrotic parts of the cartilage were gone. In their place was a mass of connective tissue cells and fibroblasts containing numerous congested blood vessels running in various directions. On each side of this mass were several bony lamellae lined by osteoblasts and attached to the lower layers of cartilage. The joint space was of course obliterated. (Fig. 15.) Still further down there was complete osseous ankylosis with here and there a few cartilage cells remaining. In this part the marrow spaces were filled with fibroblasts and congested blood vessels. In the rest of the joint there was complete osseous ankylosis. In this part the bone was somewhat denser than the surrounding bone and the marrow spaces were filled with fibroblasts, congested blood vessels, fibrin and fat cells. In the surrounding bone the marrow spaces were filled with fat cells and a few congested blood vessels. The neighboring periosteum showed slight proliferation.

In the articulation between the astragalus and the central tarsus near the interosseous ligament, there was a small area of osseous ankylosis in which the bone was denser than the surrounding bone and the Haversian canals were filled with fibroblasts. Around this area the articular cartilages were nearly normal and the surrounding bone was slightly congested. There was also a small area of fibrous ankylosis in which the fibrous tissue contained numerous fibroblasts and congested blood vessels of various sizes. Next to the bone there were areas of cartilage. Around this ankylosed area the upper layers of the cartilages were gone and the layers next to the bones were covered by granulation tissue similar to the ankylosed area. This tissue was being transformed into bone. This was shown by the presence of bony lamellae

applied to the remaining cartilage, and lined by osteoblasts. In the subchondral bones the Haversian canals were enlarged and irregular in shape. They were filled with fibroblasts, fibrin and congested blood vessels. (Fig. 16.)

In the white colored area in the center of the tibial trochlear ridge, the cartilage has entirely gone. Its place was taken by connective tissue containing some cartilage cells. The usual zones of articular cartilage were absent. At the periphery there were nests of proliferated cartilage cells. In the surrounding cartilage the upper layers showed degeneration. In the subchondral bone some of the Haversian canals were enlarged, irregular in shape and contained congested blood vessels and fibroblasts.

In the left hock the capsular ligament was slightly thickened. The synovial membrane was smooth excepting along the margins where the various bones were in apposition. Along these margins the synovial membrane was thickened by whitish fibrous tufts varying in length from 1 mm. to 1 cm., the longest being in the tibio-astragalar articulation.

There was a yellowish erosion 1.5 cm. long, 6 mm. wide and 1 mm. deep in the center of the tibial trochlear ridge. There was a similar erosion 3 cm. long, 5 mm. wide and 1 mm. deep on the corresponding astragalar articular surface. The remaining articular cartilages were roughened in places and softer than normal. They had a sort of velvety appearance.

In the calcaneo-astragalar articulation, there was an erosion 1 cm. long, 8 mm. wide and 1 mm. deep on the lateral astragalar articular facet. At the lower border of this erosion there was a growth of connective tissue. There was a partly reddish and partly yellowish erosion 6 mm. long, 2 mm. wide and .5 mm. deep on the median astragalar articular facet. There were similar erosions on the corresponding calcanean articular facets.

The articulation between the astragalus and the central tarsus was apparently normal.

In the articulation between the central tarsus and the third tarsus there was complete osseous ankylosis at the lateral and anterior aspects of the articulation, not involving the capsular ligament or the synovial membrane. In the ankylosed part, the bone was denser than the porous bone of that part but not as

dense as the cortical bone. The ankylosed area was somewhat reddened. Near the middle of the articulation the bones were partially ankylosed, some of the cartilage remaining intact. The median margin was normal but the cartilages near the median margin were roughened by shallow erosions.

The remaining articulations of the hock joint were apparently normal.

The lining of the calcanean bursa was slightly roughened throughout. On the lateral side of the tuber calcis for an area of 3 cm. x 2 cm. there were erosions surrounded by an excessive growth of connective tissue.

This is a case of a *chronic erosive, fibrous* and *osseous ankylosing osteo-arthritis* and *proliferative synovitis* as well as *chronic ossifying periarthritis.*

Case 42. An aged grey mare suffering from spavin of both hock joints. Killed by chloroform.

In the left coxo-femoral joint the articular cartilages of both the cotyloid cavity and the head of the femur were thickened, wrinkled and of the consistency of soft rubber. On section they showed fibrillation of the cartilage. The subchondral bone was apparently normal.

In both stifle joints and in the left carpal joint there were old yellowish erosions of the articular surfaces.

In both hock joints in the articulations between the astragalus and the central tarsus the articular cartilages were entirely gone from both articular surfaces. The denuded bone was yellowish smooth and apparently denser than normal except in a small area where the denuded bone was roughened and reddened. In the right hock joint there was in addition fibrous ankylosis in several areas in the articulation between the central and third tarsus.

Microscopically the surface of the denuded bone was denser than normal. Towards the center of the bone the Haversian canals were enlarged, irregular in shape and filled with granulation tissue.

All the other joints were apparently normal.

This is a case of a *chronic degenerative arthritis* and a *chronic erosive* and *fibrous ankylosing osteo-arthritis.*

Case 43. A twenty-five year old buckskin mare blind in both eyes and extremely lame on left hind limb. Killed by chloroform. There were fibrous tufts and adhesions on the pleura and the peritoneum.

In the left coxo-femoral joint there were yellowish erosions 1.5 cm. in diameter on the corresponding articular surfaces. Both stifle joints showed slight erosions.

Both hock joints were enlarged. The left was hard while the right was fluctuating. There was osseous ankylosis between the two lower rows of tarsus and the metatarsus in both hock joints. In the left tibio-astragalar articulation there was a considerable amount of reddened synovia containing flocculi of fibrin. The synovial membrane was reddened, roughened and thickened by a partly organized fibrino-hemorrhagic exudate. There was a reddish erosion 3 cm. long and 1 cm. wide in the astragalar trochlear groove. There was a reddened triangular shaped erosion on the median and an erosion 1 cm. long and 4 mm. wide on the lateral astragalar trochlear ridge. There were corresponding erosions on the tibial articulation. In the right tibio-astragalar articulation the capsule was distended by a yellowish turbid liquid that coagulated on standing. A smear showed it to contain numerous polymorphonuclear leucocytes.

Media inoculated from the left hock joint gave a growth of staphylococcus albus in pure culture. From the right hock joint there was a pure culture of B. coli producing gas in dextrose, lactose and saccharose.

This is a case of a *chronic erosive* and *osseous ankylosing osteo-arthritis* apparently inactive at this time. There is also an *acute fibrino-hemorrhagic synovitis* and *erosive osteo-arthritis* in the left hock joint and a *sero-purulent synovitis* of the right hock joint.

The cause of the long standing condition cannot be determined at this time. The acute conditions were apparently caused by infection.

Discussion

At the present time the term *spavin* stands for a *chronic ankylosing intertarsal* and *tarso-metatarsal osteo-arthritis* with or without a *chronic periarticular ossifying periostitis.* It is supposed to

originate in the subchondral bone as a *rarefying osteitis* and to spread to the articular cartilage producing erosions and ankylosis and to the periosteum producing exostoses.

In the sixty-five cases studied the pathological changes varied considerably. In the capsular ligament the early changes found were infiltration of polymorphonuclear leucocytes in two cases. In others the capsular ligaments were thickened by an oedema and hemorrhage while some showed round cell infiltration into the capsular ligament. In those of longer standing the most marked change was a thickening of the capsule by a production of connective tissue. In addition there were in this connective tissue, areas of cartilage and of bone. This is seen in the stifle joint, case 22. In one case the capsular ligament was entirely ossified (left hind ring bone, case 39) and in two instances one side of the capsular ligament was ossified (left stifle, case 16 and right hock joint, case 41).

In the synovial membrane the early changes were hyperemia and exudation. In some instances there were, in addition, petechial hemorrhages on the synovial membrane. The exudate was purulent, sero-purulent and fibrino-purulent respectively.

In many instances the exudate was mostly fibrin and in still others there was a serous exudate. In two cases there were a sero-hemorrhagic and a sero-fibrino-hemorrhagic exudate. The fibrinous exudate was in some cases free while in others it was firmly adherent to the synovial membrane. In those instances where the fibrin was adherent, it was brought about by an organization of the fibrin by connective tissue and blood vessels, arising from the synovial membrane. In one case there were areas of cartilage and of bone in this granulation tissue. Many of these were in the form of small round bodies attached to the synovial membrane by a small villous outgrowth. This suggests a possibility of one of the ways in which free bodies may occur in a joint. Such a free body was found in one case to be composed of cartilage the center of which had begun to ossify.

Of the degenerative changes observed in the synovial membrane nearly all of them were of the nature of cloudy swelling of the lining mesothelial cells. This was observed only rarely. As a rule, the changes were proliferative, instead. In several cases

these cells were loosened from their basement membranes and found detached. Necrosis of the synovial membrane was not found in these low grade forms of arthritis.

The proliferative changes of the synovial membrane were usually in the form of villous tufts varying in length from .5 mm. to 2 cm. These tufts were composed of various sized blood vessels surrounded by young and old connective tissue cells, and covered by mesothelium. Their appearance varies with the age of the process. In the early stages these tufts were small and reddened. In later stages they were longer and of a brownish color. In long standing cases they were whitish in color and composed mostly of adult connective tissue with a few blood vessels, and covered by mesothelial cells. Occasionally they were found inactive, i. e., they were the remains of a former inflammation. In these tufts there were no fibroblasts. In many of the cases the proliferation of tissue was in the form of a connective tissue network on the synovial membrane. In several instances there were masses of connective tissue forming adhesions of different parts of the synovial membrane. In three cases of short duration there were outgrowths of blood vessels from the synovial membrane forming thin reddened arborous films on the edges of the articular surfaces. Where the articular surfaces were ankylosed the synovial membrane as well as the capsular ligament were, as a rule, replaced by bone. In one case the capsular ligament was ossified while the synovial membrane was represented by a line resembling cartilage. In another case the synovial membrane was apparently not affected although there was osseous ankylosis of the adjacent joint.

While the changes of the synovial membrane were mainly exudative and proliferative, the changes of the articular cartilage were mostly degenerative. The appearance of the degeneration varied. In the early stages it manifested itself in a changed staining reaction. In this case the intercellular hyaline substance stained pinkish while the nuclei gradually became smaller until they were represented by small granules of nuclear material, which later disappeared leaving a homogeneous pinkish staining matrix. This process was seen in many instances on the articular surface while the remaining cartilage was apparently normal.

In other cases this process took place around blood vessels that budded into the articular cartilage from the subchondral bone. By this process the cartilage became vascularized. In some of the cases these two processes were present simultaneously, i. e., there was superficial degeneration of the cartilage and at the same time vascularization from the subchondral bone.

In many of the long standing cases the cartilage was thickened and softened. In some of these the consistency of the cartilage was that of soft rubber while in others, further advanced, the cartilage was soft and fibrillated. Microscopically, the cartilage was arranged in fibrils perpendicular to the articular surface. In the cells near the surface the nuclei were nearly gone. They became larger as they approached the bone, and near the zone of calcification they appeared nearly normal. This process was produced experimentally by Cresswell in 1895 by means of long maceration and with the aid of pressure. He claims that chemically these fibrils are similar to connective tissue fibres.

Necrosis of the articular cartilage appeared in the form of diffuse superficial necrosis in which the nuclei had disappeared and the matrix took a pinkish stain. This was seen in the early stages in which there was a reaction to a more severe irritation. Superficial necrosis in the form of crateriform erosions was more common. In these the cartilage had disappeared. In some of the cases the remains of the cartilage was in the form of a gelatinous mass. In others part of the cartilage was gone and the remaining cartilage was pinkish and homogeneous. In still others the cartilage was entirely gone having been apparently liquefied and absorbed. Due to the fact that articular cartilage does not regenerate very readily, it is possible that some of these are the remains of a previous inflammation.

In some of the erosions the articular cartilage was partly replaced by loose vascular connective tissue. In other erosions the cartilage was partly replaced by dense non-vascular connective tissue. In a few the cartilage was entirely replaced by what appeared to be fibro-cartilage. In the cases where there was ankylosis of the articular surfaces the cartilage was replaced by loose vascular connective tissue, by dense non-vascular connective tissue, by porous bone, and by dense bone respectively. In one case

where the cartilage was replaced by loose vascular connective tissue there was formation of bone and proliferation of cartilage in this tissue. (Right hock case 41.) In another case where the cartilage was replaced by dense non-vascular connective tissue there were areas of proliferating cartilage cells in this tissue. (Left hock joint case 37.)

The erosions in the center of the tibial trochlear ridge are designated by certain anatomists as *synovial fossae*. This would indicate that they consider them normal structures. In order to determine this point, a study was made of the joints of animals in utero as well as of older animals. In those cases designated as normal, the tibial trochlear ridges were found smooth and without a depression in the center. Some were found where the only variation from the normal were punctiform depressions on the ridge. A microscopic study of these so-called "synovial fossae" showed that they are undoubtedly pathological. Some of them show a reaction to severe irritation while others show reaction to mild irritants. There were found several in which it appeared that there was an erosion that had healed. In these healed erosions the zone of provisional calcification was absent and the cartilage resembled fibro-cartilage instead of hyaline cartilage.

In some cases there were circumscribed areas of necrosis in the center of the articular cartilage. In a few of these the necrotic tissue was a homogeneous structureless mass. In most of these the necrotic areas were calcified. After decalcification these areas showed a homogeneous hyaline substance with numerous rounded bodies resembling "corpora amylacea". (Fig. 8.) These areas are designated by Eberlein as "lime points". According to him they are areas in which the cartilage has been invaded by subchondral marrow and which later had undergone a condensing process, *i. e.*, they are areas of *ossification* in which the process had become quiescent in the stage of condensing osteitis. They show in effect one of the sequellae of necrosis. This is similar to necrosis in other tissues, particularly caseation necrosis which often becomes calcified. They do *not* contain any bone.

In the subchondral bone enlargement of the marrow spaces by hyperemia and fibrinous exudate was the most marked feature in the early stages. A fibrinous exudate with numerous fibro-

blasts and congested blood vessels were found in the enlarged Haversian canals in some cases of long standing, even in those that showed fibrous ankylosis. In two cases there were polymorphonuclear leucocytes in addition to the fibrin. Some of the enlarged Haversian canals were completely filled with endothelial cells and some were completely filled with fibroblasts. In many cases these canals were filled with fibroblasts, congested blood vessels and fibrin, and lined by osteo-blasts. In some apparently quiescent cases or those that have reached the last stages, particularly where there was osseous ankylosis, these canals were filled with fatty marrow with slight hyperemia in certain instances.

In some cases where the articular cartilage was eroded, the subchondral bone was normal. This was true in those that showed fibrillation of the articular cartilage as well as those in which there was no fibrillation. In some cases in which the erosion did not reach the subchondral bone, the latter was denser than usual. While it is possible that this was due to a condensing osteitis, this condition was not confined to the affected area so that it is more probable that the bone was naturally denser in these animals. In many of the cases the erosion affected the subchondral bone as well as the cartilage. In these instances part of the underlying bone was gone and its place was taken by granulation tissue and in one case by granulation tissue in which there were circumscribed areas of polymorphonuclear leucocytes.

In one of the cases there were foci of necrosis in the subchondral bone. These apparently began immediately under the cartilage and spread towards the center of the bone. The overlying cartilage was degenerated but intact.

Eburnation of the denuded articular ends of bones was seen in two instances. In the left stifle joint, case 16, and in both intertarsal articulations, case 42. Eburnation is described as occurring in freely movable joints. In the latter case the articulations between the central tarsus and the astragalus were affected. This joint is nearly devoid of motion.

In most of the cases there were no apparent changes in the periosteum. In some of them the periosteum was slightly thickened by a proliferation of large bluish staining cells arranged in rows parallel to the fibres of the attached ligaments. In one case the

periosteum was greatly thickened by connective tissue and in two of the cases there was an ossifying periostitis.

To summarize the changes, the final stage of the process is an *osseous ankylosis* of the two adjacent bones. This process may be confined to the joint or it may be accompanied by a periarticular ossification. This is, in many cases at least, preceded by a *fibrous ankylosis*. The fibrous tissue may be dense, non-vascular with or without areas of proliferating cartilage. It may also be loose, rich in fibroblasts, and richly supplied with blood vessels. In one case there was beginning ossification in this tissue. This form of ankylosis in some instances aparently began in the joint by an organizing exudate. The proliferation of tissue apparently began in the synovial membrane or around the interosseous ligament. In most cases it was impossible to tell where this form of ankylosis began. Preceding this stage there is erosion of the articular cartilage with or without involvement of the subchondral bone. These erosions began in the subchondral bone in some cases. In others it undoubtedly began in the joint sinus. In those that involved the bone as well as the entire depth of the articular cartilage, it was impossible to determine the point of origin. The proliferation of the synovial membrane may start at any period of the process. The formation of villous tufts on the synovial membrane is so common that they have been considered as normally occurring on the synovial membrane. They are designated by some authors as " synovial villi ". In this study normal synovial membranes have been encountered in which the surface was whitish, smooth and shiny, similar to that of other serous membranes. The early changes are hyperemia and exudation on the synovial membrane, hyperemia and exudation in the marrow spaces with widening of these, and degeneration of the articular cartilage. These changes may take place at the same time or they may occur individually. The process may become quiescent at various stages.

The synovial membrane is the only structure in the immediate proximity of the joint sinus that is supplied with blood vessels. This makes the synovial membrane more resistant to irritation than the non-vascular articular cartilage. Regeneration of the articular cartilage does not take place very readily, while all the

structures of the synovial membrane regenerate with ease. For these reasons the same causative agent that would stimulate the synovial membrane to proliferation might produce degeneration and necrosis of the articular cartilage. The transition between the synovial membrane and the articular cartilage is gradual, so that there are blood vessels at the edge. This may account for the occurrence of the erosions a few millimeters from the articular border. Around the extreme articular border some of the cases studied show proliferative changes similar to those found on the synovial membrane.

Practically nothing new can be advanced regarding the point of origin of spavin, since it has been ascribed to nearly every structure encountered in the neighborhood of the hock joint. It was thought to arise from the ligaments, bursae, tendons, and the synovial membrane around the hock joint. It was even thought that it originates from a gonitis. Finally it was believed to originate from the articular cartilage and from the subchondral bone. Since Havemann's discovery of the erosions in the joint, the speculations centered around the question whether the development of spavin was from within outwards, or from the outside inwardly. Beginning with Gotti and affirmed by most veterinarians of today, the former view was considered the correct one.

In these cases, with three exceptions, all the bursae were apparently normal. In those that showed pathological changes, they were not of longer standing than the changes in the joint. It is undoubtedly possible that a traumatic injury of any kind may produce an ossifying periostitis which may later spread to the synovial membrane and then to the joint. There is in this laboratory a dry specimen of a case of an ossifying periostitis not involving the joint. The coronary ring bone of the right fore leg in case 37 and that of the right hind leg in case 39, also illustrate this point. There are, however, numerous cases of spavin in which the periosteum is not involved, and when involved shows changes that are less marked than those in the other structures of the joint. This is illustrated in many of the cases described in this paper which shows conclusively that certainly not *all* cases of spavin originate in the periosteum. In many of the cases there were degeneration and erosions of the articular

cartilage together with proliferation of the synovial membrane, without any affection of the bone. In these, the zone of calcification was intact. In some of the cases the erosions began by a fibrinous exudate in the joint sinus. In these instances the point of origin was undoubtedly in the joint and not in the subchondral bone. In some of the cases the erosions began by vascularization of the articular cartilage. These blood vessels emanated from the subchondral bone. This makes it possible that in some instances the point of origin is in the subchondral bone. In one case there were necrotic foci in the subchondral bone while the articular cartilage showed degeneration. It seems that here the origin was in the subchondral bone.

The erosions in these cases are on opposing articular cartilages. This is explained more readily on the assumption of an intra-articular irritation, than as a subchondral irritation. Nathan holds that these are due to subchondral infective foci. This would mean that the foci were by chance in corresponding areas of the opposing bones. The cases where the erosions are exactly opposite each other are too numerous to be due to chance. It is possible that an irritant on the surface of the articular cartilage may be sufficiently severe to call forth a vascularization of the cartilage from the subchondral bone. If this is so, it is possible that many erosions that appear to begin in the subchondral bone are in reality caused by an intra-articular severe irritant. Such an irritant on the cornea, particularly a bacterial irritant, will produce vascularization of the cornea. The cartilage is likewise a non-vascular tissue, and it may react to a severe irritant in the same manner as the cornea. The erosions around the interosseous ligaments seem to indicate that the irritant producing them is in the joint proper.

Since there are various forms of inflammations in the hock joint that may eventually lead to ankylosis and exostosis, it seems advisable to have the term *spavin* indicate any inflammation of the hock joint that may lead to ankylosis and exostosis. The meaning of the term spavin has been previously changed. As it stands today it does not include severe irritation in the joint which later terminates in ankylosis and exostosis.

That the erosive forms of arthritis in the hock joint are

included in the term *spavin* is evidenced by the various descriptions of this condition, beginning with Havemann's finding of these erosions. It seems advisable to include the exudative forms of arthritis in the hock joint, since they too may lead, and often do lead, to ankylosis and exostosis.

The present views concerning the etiology of spavin are not entirely satisfactory. With very few exceptions, mechanical irritation is considered as being the cause of spavin. It is thought to be brought about by pressure on the articular ends, stretching of tendons and ligaments, etc. The erosions in spavin are described as being punctiform in the early stages. It seems doubtful that pressure in a joint should be concentrated in an area a millimeter in diameter. In those cases where there was fibrillation of the articular cartilage, there are experimental data to show that it might be produced by pressure. Even here, however, pressure alone does not seem to be sufficient to produce such changes. Traumatism is a satisfactory explanation only in those cases that apparently began with a periostitis or an open joint.

Considering the frequency of arthritis following diseases of other organs notably acute pneumonia, pericarditis, pleuritis, peritonitis, an ulcerated tooth, or other suppurating foci, it seems possible that the true cause of many a lameness is overlooked. The history is very often unsatisfactory. The lameness is often ascribed to a sprain where it may rather be a sequel of a forgotton inflammatory condition. Organisms may get into the circulation and finally lodge in the joints without producing any local lesion elsewhere in the body. The joints not being richly supplied with blood are favorable seats for multiplication of microorganisms.

Spavin is more common in some localities than in others. Here in Ithaca spavin is a common affection of horses. This has been ascribed to the hilly country. Dr. W. L. Williams claims that during his practice in the foothills of the Rocky Mountains spavin was rarely encountered. Certainly the Ithaca hills then cannot be the cause of the frequency of spavin.

Various authors claim that spavin is more common in young than in older animals. According to the experiences of Drs. W. L. Williams and J. N. Frost, spavin is most common in horses between the ages of two and four years. The reason for this

fact advanced by many authors is the nervous temperament of younger horses. It is because "they attempt their work more vigorously than older animals". This explanation does not seem to be very satisfactory. It seems possible that the reason spavin is less common in older animals is because of *age immunity.* Younger animals generally are more susceptible to infection than older ones. The nervous temperament may be a contributing factor.

Many of these cases were undoubtedly caused by intra-uterine or umbilical infection. The variation of the lesions in these cases was apparently due to the variation of the resistance of the animals, the variation of the virulence of the cause perhaps, and the variation in the ages of the processes. Case 22 is a notable example of well advanced lesions caused by umbilical infection. A case similar to this one has been observed. It was about the same age with a similar clinical history. A veterinarian ordered the destruction of the animal and the order was not followed. The animal recovered sufficiently to give eight years of valuable service. The dry specimen showed erosions in the right stifle and a spavin with articular and periarticular ankylosis of the left hock joint. It was impossible to make a thorough examination of these joints because the limbs were mounted.

In both these cases the cause was apparently local infection resulting from umbilical infection. They show that it is possible for spavin to develop from umbilical infection.

Schofield in his study of joint-ill in foals concludes that it is closely associated with a hemolytic streptococcus in which case the infection is probably by ingestion, since these organisms were found in the milk of the dam. Other means of infection are, according to him, intra-uterine, and through the unhealed navel. He illustrates cases that recovered and those that resulted in ankylosis.

Dr. W. L. Williams cites a case of a herd in which there were about fifty suckling foals. Many of the foals suffered from spavin, ring bone and allied diseases. This herd was pasturing in a swamp. When the pasture was changed to dry land, spavin ceased to exist in the herd. This case apparently corresponds to Saunier's "l'eparvin gras".

In these foals running about freely in a pasture the cause does not seem to be in stretching of tendons, overexertion, heavy loads or even excessive pressure on the cartilages. On the moist pasture infection seems to be the more likely cause of the spavins. The fact that it disappeared from the herd after change of pasture would strengthen this view.

In many of these cases the pathological changes are similar to those caused by infection. A marked fibrinous exudate, for example, is as a rule caused by infection. In many of these cases this is the most conspicuous change. The villous tufts on the synovial membrane are similar to those commonly occurring on the peritoneal or pleural surfaces. In the latter cavities they are thought to be due to bacterial irritation, so that in the joint they are possibly of an infectious nature.

In many of the cases studied bacteria were found in the joints. In some they were obtained in pure culture while in others there were mixed infections. In several instances where culture media inoculated from other organs of the body remained sterile, a growth was obtained from the joints. The various bacteria isolated were B. coli, staphylococci, streptococci, an unidentified rod-shaped organism in one case, and Bact. pyogenes in one case in a cow with suppurative mastitis in addition to joint lesions.

Lesions similar to most of those found in the cases studied were produced experimentally by Nathan. They were produced in dogs by intravenous inoculations of hemolytic streptococci, pneumococci and staphylococci respectively. In this series all the organisms produced practically the same changes, so that these lesions are not those due to a specific cause.

In a recent study of spontaneous chronic arthritis in swine, Sekiguchi and Irons found hemolytic streptococci in two, bacilli in nine, and a mixed infection of streptococci and bacilli in four cases. In two they found bacteria in smears but not in culture, and in two they found no bacteria. They produced joint lesions in pigs by subcutaneous and intravenous inoculation of the organisms found.

These experimental data seem to strengthen the belief that many of the cases studied were caused by infection.

From a study of these cases the following conclusions seem justifiable.

1. That there are very commonly found in horses and cattle low grade polyarticular inflammations, leading to erosions and ankylosis.

2. That in these cases the hock joints are the ones most often affected by these inflammations.

3. That it is advisable to have the term spavin indicate any inflammation of the hock joint leading to ankylosis.

4. That the origin of spavin may be in the joint proper as well as in the subchondral bone.

5. That in many cases, spavin is apparently caused by infection.

6. That the primary seat of erosions and ankylosis may be in any of the intertarsal as well as the tarsometatarsal joints.

7. That the so-called "synovial fossae" are pathological erosions.

8. That the so-called "synovial villi" are, in many cases at least, pathological outgrowths caused by mild irritation.

9. That the so-called "lime points" in the articular cartilages are areas of necrosis in which calcification has taken place.

10. That a periarticular ankylosis is, in some cases at least, due to an ossifying inflammation of the capsular ligament.

11. That in the cases studied, osteoclasts do not play an important part in the rarefying osteitis.

[The author wishes to take this opportunity to thank Prof. S. H. Burnett, under whose supervision this work was done, for his encouragement and helpful criticisms. I am also indebted to him for selecting this subject, and for the gross description of Cases 17 and 37.]

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PLATE No. I.

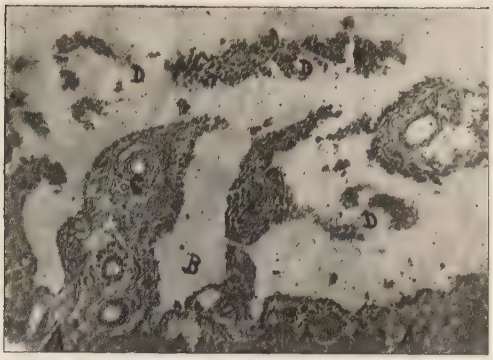
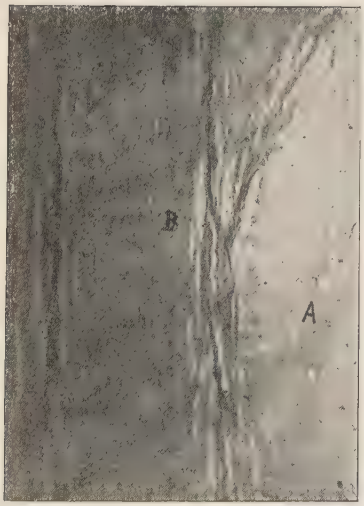


Fig. 1.



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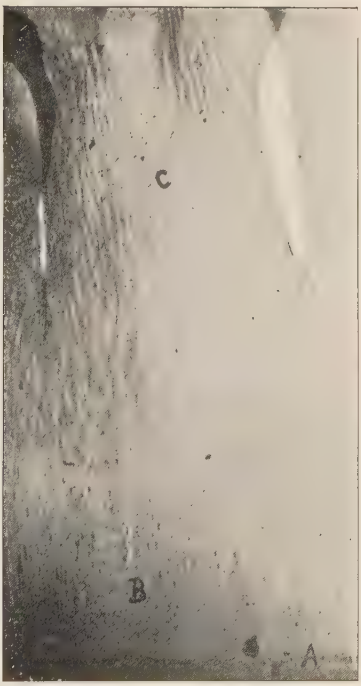

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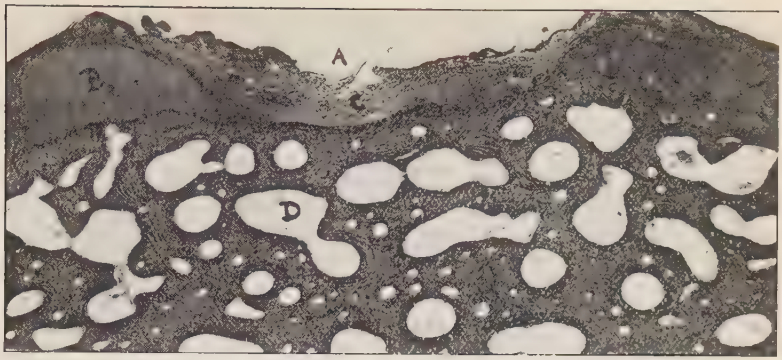

FIG. 4.

FIG. 5.

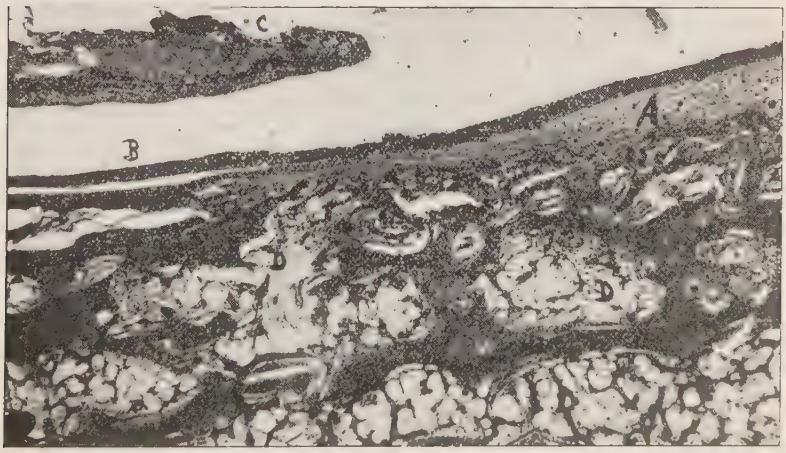

FIG. 6.

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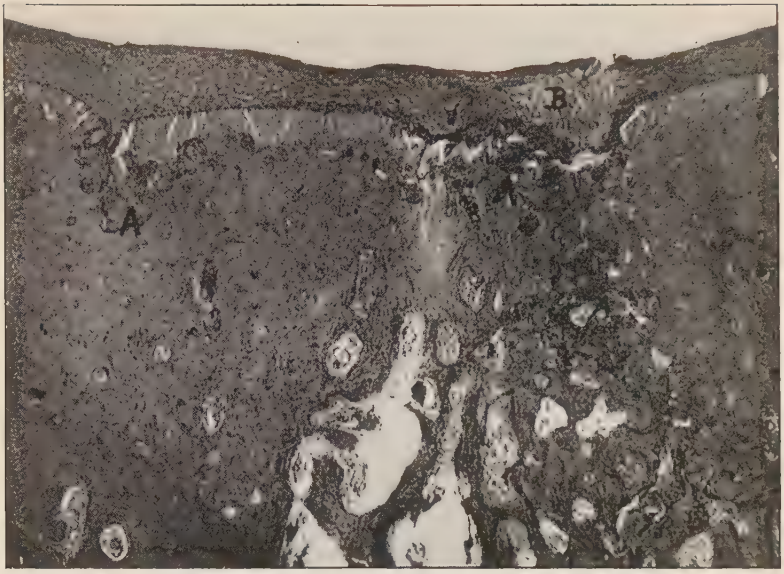

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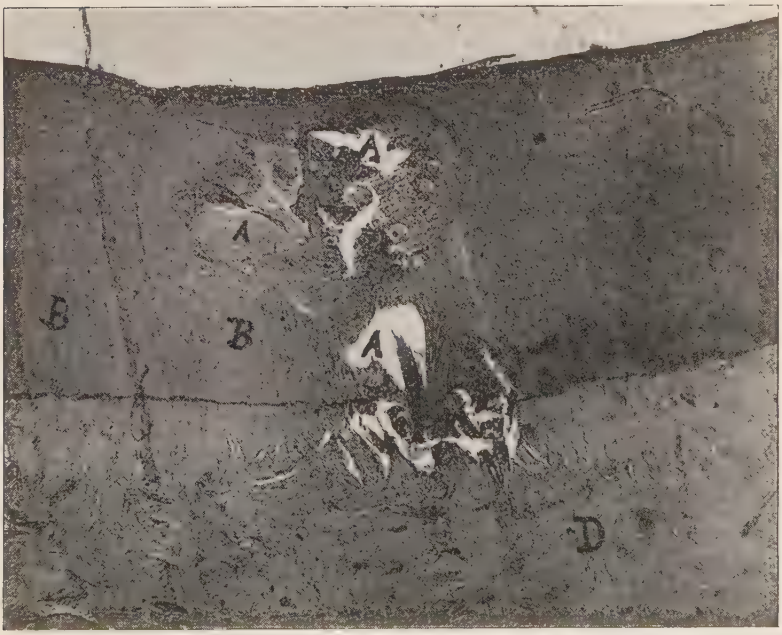

Fig. 8.

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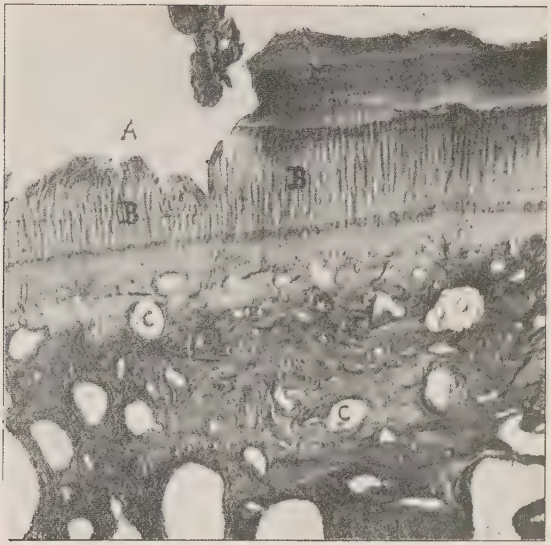

Fig. 9.

Fig. 10.

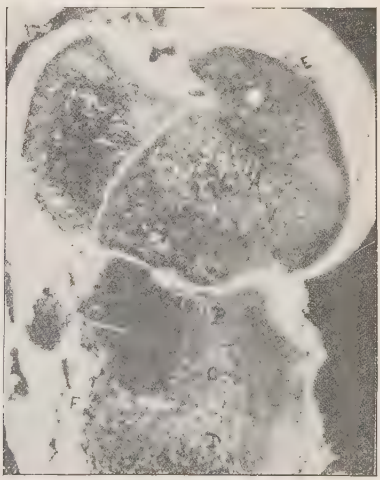

Fig. 11.

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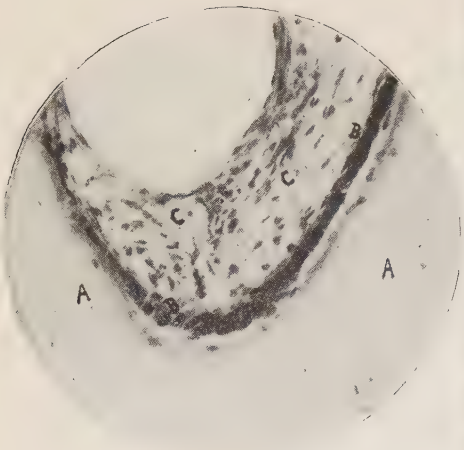
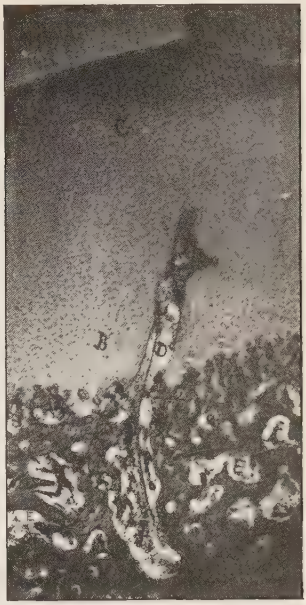


FIG. 12.



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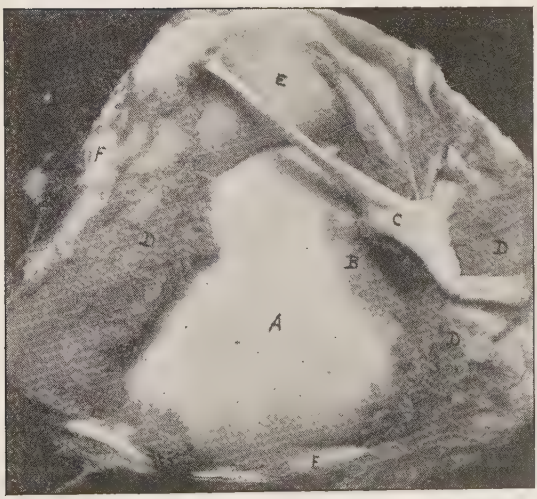

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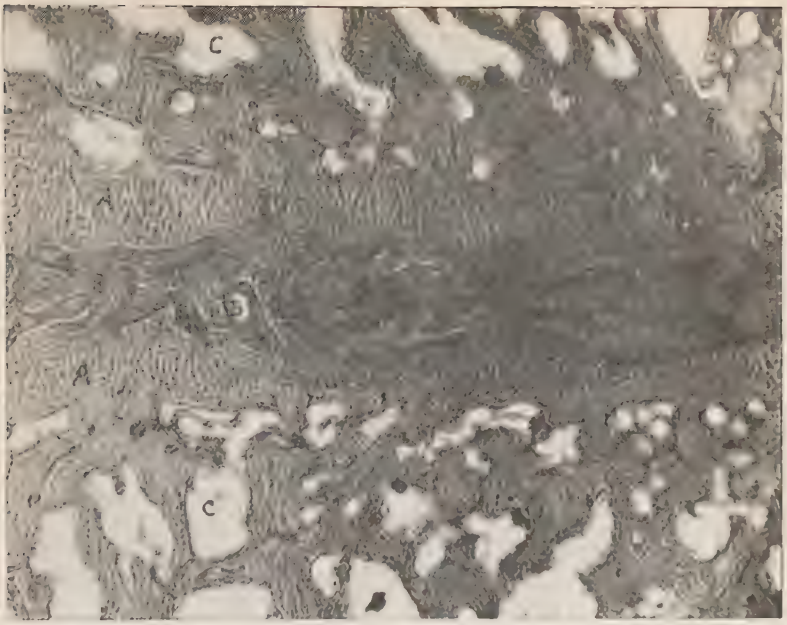

Fig. 15

Fig. 16.

PLATE No. VII.

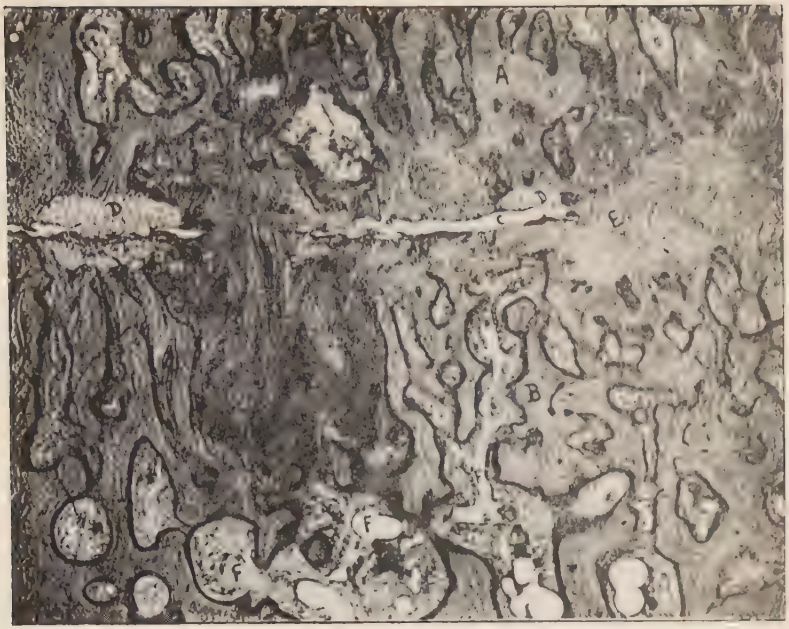

Fig. 17.

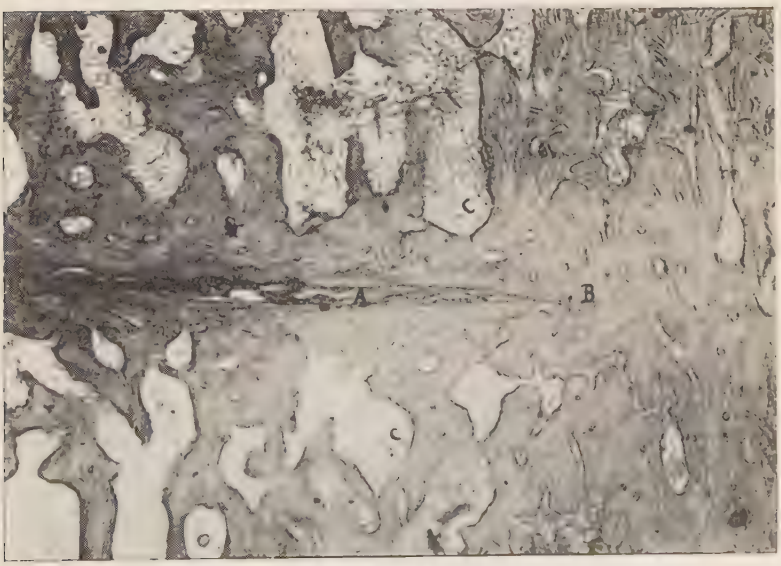

Fig. 18.

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Woodhead, G. S. Practical Pathology. London, 1910.

Zalewsky. Die Gonitis chronica deformans des Pferdes. *Monatshefte f. prakt. Thierheilk.* XII Bd. (1901).

Description of Plates

Plate I

Fig. 1. Desquamation of synovial membrane. Case 1. x 100.
- A. Capsular ligament.
- B. Villous tufts covered by mesothelial cells.
- C. Blood vessels in the villous tufts.
- D. Desquamated masses of mesothelial cells.

Fig. 2. Fibrillation of semilunar cartilaginous disc. Case 1. x 100.
- A. Group of remaining degenerated cells.
- B. Fibrils in place of the cartilage.

Fig. 3. Fibrillation of articular cartilage proximal end of tibia. Case 1. x 45.
- A. Zone of provisional calcification.
- B. Nearly normal cartilage cells.
- C. Fibrillation of cartilage cells.

Plate II

Fig. 4. Erosion originating in the joint. Right tibial trochlear ridge. Case 49. x 20.
- A. Area where cartilage is gone.
- B. Remaining articular cartilage.
- C. Pinkish staining homogeneous material in place of cartilage.
- D. Normal subchondral bone.

Fig. 5. Erosion of median trochlear ridge right astragalus. Case 22. x 20.
- A. Articular cartilage nearly normal.
- B. Fibrous connective tissue in place of cartilage.
- C. Fibrous connective tissue in place of bone.
- D. Blood vessels from the subchondral bone invading the cartilage.

E. Rarefied subchondral bone.
F. Area where cartilage is absent.

Fig. 6. Erosion of astragalar trochlear groove with intra-articular exudate. Case 7. x 30.
A. Remaining articular cartilage.
B. Area where cartilage is gone.
C. Intra-articular exudate of fibrin containing erythrocytes.
D. Enlarged subchondral marrow spaces filled with fibrin and congested blood vessels.

Plate III

Fig. 7. Subchondral foci of necrosis right central tarsus. Case 30. x 30.
A. Foci of necrosis.
B. Degenerated articular cartilage.

Fig. 8. Calcified area of necrosis in the articular cartilage tibio-astragalar articulation. Case 57. x 40.
A. Calcified area of necrosis.
B. Degenerated articular cartilage.
C. Articular cartilage nearly normal.
D. Subchondral bone denser than normal.

Plate IV

Fig. 9. Erosion originating in the joint. Left astragalar trochlea. Case 35. x 20.
A. Area where cartilage is absent.
B. Fibrillation of the remaining articular cartilage.
C. Subchondral bone apparently normal.

Fig. 10. Fibrous ankylosis antero-posterior section of right hock joint. Case 41. One-half natural size.
A. Calcaneum.
B. Astragalus.
C. Central tarsus.
D. Third tarsus.
E. Principal metatarsus.
F. Interosseous ligaments.
G. Ankylosis of the calcaneo-astragalar articulation.
H. Area where this articulation is not ankylosed.

I. Area of ankylosis in the articulation between the astragalus and the central tarsus.
J. Normal articulations.
K. Thickened capsular ligament.

Fig. 11. Osseous ankylosis, right hock joint. Case 38. One-half natural size.
A. Upper row of tarsus.
B. Central tarsus.
C. Third tarsus.
D. Metatarsus.
E. Erosions in articular cartilages.
F. Areas where joint is not ankylosed represented by white lines.

Plate V

Fig. 12. Enlarged Haversian canal. Left third tarsus. Case 37. x 250. Taken from same specimen as Fig. 17.
A. Bony lamellae.
B. Osteoblasts lining the Haversian canal.
C. Haversian canal containing fibrin and fibroblasts.

Fig. 13. Thickened area on articular cartilage with beginning erosion from the subchondral bone. Right tibial trochlear ridge. Case 5. x 29.
A. Thickened area on articular cartilage.
B. Normal articular cartilage.
C. Degenerated articular cartilage.
D. Beginning erosion of the articular cartilage by vascularization from the subchondral bone.
E. Congested subchondral bone.

Fig. 14. Adhesions of different parts of the synovial membrane. Left patellar articulation. Case 16. 9/10 natural size.
A. Patellar articular surface.
B. Arborous film of blood vessels on the articular cartilage.
C. Band of connective tissue forming an adhesion between several parts of the synovial membrane and the patellar articular surface.
D. Filliform and papilliform villous tufts on the synovial membrane.

E. Accessory cartilage of the patella.
F. Thickened capsular ligament.

Plate VI

Fig. 15. Fibrous ankylosis that apparently began in the joint. Right calcaneo-astragalar articulation. Case 41. x 30.
A. Remaining cartilages on both articular surfaces.
B. Embryonal connective tissue and blood vessels in the joint sinus.
C. Subchondral bones.

Fig. 16. Fibrous ankylosis in the articulation between the astragalus and central tarsus right hock. Case 41. x 75.
A. Articular cartilage nearly normal.
B. Fibroblasts and blood vessels producing ankylosis of the joint.
C. Proliferating fibrocartilage in the ankylosis.
D. Formation of bone in the ankylosis.
E. Enlarged Haversian canals containing fibrin, fibro-blasts and congested blood vessels and lined by osteo-blasts.
F. Degenerated articular cartilage.
G. Exudate of fibrin in the joint sinus.

Plate VII

Fig. 17. Fibrous ankylosis of left tarso-metatarsal joint. Case 37. x 25.
A. Osseous tissue third tarsus.
B. Osseous tissue metatarsus.
C. Articular cavity.
D. Remaining articular cartilage.
E. Fibrous ankylosis.
F. Enlarged Haversian canals filled with fibrin, fibroblasts, congested blood vessels and lined next to the bone by osteoblasts.

Fig. 18. Osseous anklosis of right tarso-metatarsal joint. Case 38. x 20. Taken from same specimen as fig. 11.
A. Exudate in articular cavity.
B. Osseous ankylosis.
C. Fatty marrow in enlarged Haversian canals.

PARASITES AFFECTING SHEEP

C. P. Fitch
Department of Comparative Pathology and Bacteriology

The losses from disease among sheep in this State (New York) are largely the result of infestation by some animal parasite as worms or ticks. Infectious and non-infectious diseases of other than parasitic origin are relatively uncommon here. At this time when the sheep raising industry is being stimulated and the farmer is being urged to increase his flock or to begin keeping one it is thought well to present this necessarily brief and, as far as possible, non-technical report on the common parasitic diseases of this species of domestic animal. Further, in the post-mortem work done at the college during the past year in the regular teaching routine, the author has been thoroughly impressed with the great economic importance of parasitic disease among sheep and especially that due to the stomach worm (*Haemonchus contortus*). This is not a new disease as it is known to occur in nearly every locality where sheep are raised. We simply wish to impress upon the veterinarians of this State the importance of this disease and its high frequency.

It is especially true in dealing with animal parasites that in the majority of cases after a flock once becomes infested with a parasitic disease it is very difficult to bring about a cure. The real "key note" is to prevent the animals from becoming infested and this can only be brought about by a knowledge of how and where the sheep come in contact with the particular parasite. Many of the parasites pass one stage of their life cycle on one species of animal and then are transferred to another species to complete their development. It is when these facts are understood and appreciated that satisfactory methods can be undertaken to protect a flock from the ravages of parasitic disease. With all the study and extensive researches that have been carried out there remain many things which are still unknown concerning the life of some of the parasites. In this report the aim will be to discuss the common parasitic diseases, first, as to *cause* (a brief description including the life cycle of the parasite), second, *symptoms* produced and whereby a positive diagnosis may be made, third, *treatment* and fourth, *prevention*.

EXTERNAL PARASITES

Those affecting the skin and nasal chambers.

SHEEP TICK. (*Melophagus ovinus*).

Description. This is one of the most common parasites of sheep. It is not in the technical sense a tick although commonly spoken of as such. It is really a "louse fly". Its complete life cycle is spent on the sheep and therefore it is much less complex than that of the true tick. The sheep-tick is more like a fly than a true tick. It possesses, as do flies, six legs while true ticks have eight. Zoologists regard it as a kind of fly that has lost its wings. The parasite needs no wings as it remains for its whole life on the sheep. The adult tick is ⅛ to ¼-inch in length and is reddish or grayish brown in color. The head is small and sunken into the prothorax. The abdomen is rather large and oval in shape.

The whole life of the tick is spent on the sheep. They will not live more than a few days off their host. The egg undergoes some development within the body of the female. It is laid as a larva which changes into the pupa stage in about twelve hours. During the summer months the pupa requires nineteen to twenty-three days to hatch. In colder weather this period is lengthened up to forty to forty-five days. The females reach sexual maturity in from fourteen to thirty days.

The ticks, while feeding, attach themselves for the time being to the skin of the sheep by means of their sucking tubes; when they are not feeding they move about in the wool.

Sheep that are heavily infested with these parasites scratch or dig themselves upon nearly every object which avails itself for the purpose. If the ticks are causing trouble they can readily be found by examining the skin of the animal by parting the wool. The parasites are large and are readily observed with the unaided eye. The injury caused by these parasites is not only in the loss of wool but more especially in the flesh of the animal itself. A heavy infestation leads to malnutrition and among lambs often to the death of the individual. The lambs will not grow and remain stunted if not relieved of the vast numbers of the "tick".

Treatment. The ticks can be greatly lessened by the vigorous use of *pyrethrum,* a dusting powder, and the remedy is available

during cold weather. But by far the most practicable method and the one that gives the best results is the use of "dips". A large number of different parasiticides are used and the one to be selected must be best adapted to conditions. Among these should be mentioned the coal tar products such as creosote, creosol, kresco, etc., and the nicotin dips. "The only home made dip which has proven efficacious is the lime-sulphur-arsenic dip. This dip will eradicate sheep ticks with one dipping when all conditions are favorable, but one dipping cannot be depended upon in practical operations."

The following account of the manufacture and application of this dip is taken from the bulletin by Imes:

"The lime-sulphur-arsenic dip is made by mixing standard strength lime-sulphur dip with one-half standard strength arsenical dip. Directions for making lime-sulphur dip and the arsenical dip and mixing the two to form a dip for sheep ticks are as follows:

"The lime-sulphur dip is made in the proportion of eight pounds of unslaked lime (or eleven pounds of commercial hydrated lime, not air-slaked) and twenty-four pounds of flowers of sulphur or sulphur flour to 100 gallons of water. Place the lime in a water-tight, shallow box and add sufficient water to form a thin paste. Sift the sulphur into this and mix well until a paste of about the consistency of mortar is formed, adding water as required. Place this lime-sulphur in thirty gallons of boiling water and boil for one hour, adding water from time to time to maintain the quantity at thirty gallons or in that proportion. During the boiling process the mixture in the boiling tank should be stirred well to prevent the paste from settling and caking on the bottom of the tank; the boiling process should be continued until all sulphur disappears from the surface. A large mortar hoe is a good implement with which to stir the boiling mixture. The lime and sulphur should both be weighed; do not trust to measuring them in a pail or guessing at the weight. It sometimes happens the sulphur is not all "cut" or dissolved; this is especially true if the lime is not first-class quality. Small quantities of extra lime may be added if during the cooking it is noted that the sulphur is not being "cut" properly. As an excess of lime in the dip will tend

to injure the sheep and the wool, no more lime should be added than is necessary to cut the sulphur. After the mixture has been boiled for 1 hour the liquid should be of a chocolate or dark amber color.

" The contents of the boiling tank should be drawn off or dipped out and placed in the settling tank and allowed to stand until all sediment has settled to the bottom and the liquid is clear. The use of some sort of settling tank provided with a bunghole is an absolute necessity, unless the boiler is so arranged that it may be used for both boiling and settling. An ordinary water-tight barrel will answer very well for a settling tank at small vats. All settling tanks of every nature should have an outlet at least four inches from the bottom, in order that the clear liquid may be drawn off without becoming mixed with any of the sediment. Drawing off the liquid as above indicated has an advantage over dipping it out, for the reason that in the latter case the liquid is stirred more or less and mixed with the sediment. The prime object is to get the clear liquid without any sediment. The sediment should, under no circumstances, be allowed in the dipping vat, as it will injure the wool and the eyes of the sheep.

" When the sediment has fully settled, draw off the clear liquid into the dipping vat and add warm water sufficient to make a total of 100 gallons of dip. When mixed and cooked as above specified the concentrate is three and one-third times the strength required for the dip in the vat, so that to every thirty gallons of such concentrate seventy gallons of warm water should be added to make a dip of the required strength.

" In preparing lime-sulphur dip in large quantities, several hundred gallons of concentrate are often made at one time in a single large cooking tank. The quantity made at one boiling is limited only by the facilities at hand. If the boiling tank is of sufficient capacity enough lime-sulphur paste should be cooked at one time to dip the flock. The quantity of mixture in the cooking tank may be varied at will, but the proportions of the various ingredients should not be altered.

" To each 500 gallons of lime-sulphur solution prepared and diluted as above directed add four pounds of arsenic and twelve pounds of salsoda made into a solution as follows: Put twelve

gallons of water in a kettle or tank, heat to boiling, and add twelve pounds of salsoda; when this has been dissolved add four pounds of powdered white arsenic, then boil and stir for fifteen minutes or longer, until the white arsenic has entirely disappeared. The quantity of arsenical solution prepared at any one time is limited only by the capacity of the kettle or tank, but the proportions of the ingredients should not be altered. The arsenical solution should be added to the diluted lime-sulphur solution in the vat. When the arsenical solution is added a yellow-colored flocculent precipitate is formed which remains in suspension. The liquid in the vat should be well stirred before dipping operations are commenced. This dip can be used in any kind of water without injury from the water, but there is no field test for its strength.

"*It should be remembered that this dip is poisonous, and due precaution should be taken in handling and using it.* The sheep should be held in the draining pens and holding corrals until all surplus liquid has drained from the fleeces. When the vat is emptied the dip should be disposed of in such manner that the animals may not have access to it. Preferably it should be run into an inclosed pit."

For more detailed information regarding the eradication of this parasite the following are recommended: The Eradication of the Sheep Tick, Bulletin No. 105, University of Wyoming Agricultural Experiment Station, Laramie, Wyo.; Farmers' Bulletin, 798, The Sheep Tick and its Eradication by Dipping, United States Department of Agriculture, Washington, D. C.

Sheep Scab

This is one of the most serious as well as one of the oldest diseases affecting sheep. It is a highly contagious skin disease and is easily transmitted from one animal to another. It is a fortunate thing that this disease is not widespread in this State, but it is quite common in the west and central west and with the present importation of sheep from these regions its dissemination is to be feared. The veterinarian should be on his guard against the introduction of this disease into the flocks of his clients. When allowed to spread, this disease causes serious

financial loss to the industry. The principal losses result from (1) death of the infested animals, (2) general debility, malnutrition and loss in weight, (3) loss in the quantity and quality of wool produced.

Cause. The parasite which causes this disease is very small. When full grown the female is only about 1/40 of an inch and the male 1/50 of an inch in length. They may, however, be seen with the unaided eye if they are placed on a dark background. A low power hand magnifier is, however, a great aid in the recognition of this parasite. Its technical name is *Psoroptes communis* var. *ovis.*

To make a positive diagnosis of the presence of this parasite in the flock it is simply necessary to make some scrapings from an infested area and place on a dark piece of paper. The very small mite can be seen crawling about if carefully observed. As stated above a hand magnifier is a great aid in this diagnosis.

The various stages in the life history of this mite are all passed on the body of the sheep. The females deposit their eggs in masses at the base of the fibers of the wool. They hatch after three or four days' incubation and the young mites grow to maturity in seven or eight days. The females live but a short time after they have laid their eggs. According to Gerlach as many as 1,500,000 mites may result as the progeny of a single pair of mites in ninety days. After another fifteen-day interval the number may be as high as 15,000,000. These figures, although theoretical, serve to show how rapidly the parasite breeds and how even a pair of mites may in a short time bring forth enough parasites to affect the whole flock. It can readily be seen that prompt treatment is essential.

Symptoms. The following account of the symptomatology of this disease is taken from Law, Veterinary Medicine, Vol. V.

" The affected sheep attracts attention by the rough state of its fleece at the affected part, the wool being flattened or ragged, with white tufts rubbed out or pulled out by the teeth. It shakes its tail, rubs its body, scratches with its hind feet, and tears with its teeth in a way to show intolerable pruritus. If the affected parts are rubbed with the hand the patient shows its enjoyment by the scratching movements of the hind limbs, the trembling and

nibbling with the lips, the movements of the head and body as if rubbing. The itching is increased by driving or by crowding together so as to heat the sheep and induce perspiration. The skin of the affected part is covered with yellowish papules of varying size, and a marked accummulation of scurf among the roots of the wool. Later the affected skin swells uniformly, and the increasing exudation concretes into a massive scab enveloping the roots of the wool, so that as it increases layer by layer on its deeper surface, it lifts the fibres out of their follicles, detaching the wool and leaving extensive bare scabby patches. The denuded surface shows all the variation of lesions shown in other mangy animals. Papules, vesicles, pustules, scabs, cracks, excoriations, and even sloughs may appear at different points. Sometimes in clipped sheep the exudate forms a uniform, smooth, parchment-like crust covering the whole exposed area. Around these bare patches the wool is encrusted at its roots, or shows a dark, dirty, scurfy layer composed of epidermic cells, yolk, dried exudate and the exuviae of the acarus. Beneath this the parasite is found in myriads. The bare spots may show comparatively few. The intense itching of these parts and the readiness with which the wool can be pulled out are significant.

"Unlike the sarcoptes and symbiotes, the psorptes attacks by preference the parts that are thickly covered by wool, and generally respects those having a hairy coat only. For the same reason it is especially inveterate on fine wooled sheep, the density of the wool and the abundance of yolk furnishing a better protection than in the long wooled. In all, the affection is to be looked for primarily along the back, shoulders, neck and tail, then on the sides of the body, arms, haunches and thighs, and not at all on the bare face, belly, inner sides of the limbs and lower surface of the tail. Another noteworthy point is that lambs which have a good covering of wool are liable to be severely attacked just after the clipping of their dams, the parasites migrating in numbers to the animal which furnishes the greatest measure of protection.

"The changes in the wool, in old standing cases are marked. It may be dry, rigid and lustreless from being cut off from its source of nourishment; it may be thickened at both ends and

thin in the centre of the fibre, the latter corresponding to the period of extreme cutaneous congestion and impaired nutrition; or it may be composed throughout of fibres of unequal length, the short being those that have started since the irritation has lessened. These conditions of the wool may result as well from variable feeding — rich and poor — or good or bad general health, so that they must not be looked on as peculiar to scabies.

"As in all cases of violent local irritation and infection, the subjacent lymph glands may become enlarged and tender, and suppurating and septic foci may form in distant organs.

"Left to itself the disease tends to constant advance, with increasing emaciation and debility, and leading to marasmus and death. It makes less progress in the open summer pasture than in close, warm buildings, but more in the very young or in animals that are emaciated and weak than in those that are in the prime of life, and in the fine-wooled than in the long-wooled. In exceptional cases, it is claimed that long-wooled sheep, in middle age, and in high condition, on rich pastures will get well spontaneously, but in any case they respond much more readily to judicious treatment than do others. This tendency of the disease to stand still or even abate somewhat when at pasturage, accounts partly for the partial or complete neglect of scabby sheep kept on open ranges and rarely or never shut up indoors."

Treatment. The only efficient way to treat this disease is by dipping. This has been partially described under treatment of "ticks." Two dippings ten to fourteen days apart are necessary to affect a cure. One of the most efficient as well as cheaply prepared dips is that previously described (p. 343), the lime-sulphur-arsenic preparation. A great variety of apparatus is in use for the application of dips but for a small flock an ordinary wooden box (which is made water proof) 4 feet long at the bottom, 2 feet wide and 3 feet deep and having an inclined plane extending up from the bottom at each end furnished with cleats to give a foot hold. Two men dip a sheep in the bath back downward and hold by fore and hind limbs and neck. The greatest care must be taken to prevent the head getting under the surface as a single swallow of the liquid will prove fatal. The "dip" should be worked into the wool and skin for three minutes and

this is favored by having the solution warm, about 80° F. The sheep is than laid on the inclined plane and the wool wrung out to free of the liquid which runs back into the tub. Great care must be taken not to turn the recently dipped sheep into a pasture where there is grass as often enough of the liquid will drain off from the animal to poison the forage. For more detailed information concerning the application of dips on a large scale the veterinarian is referred to Farmers' Bulletin No. 713. Sheep Scab., United States Department of Agriculture, Washington, D. C.

Prevention. As this is an exceedingly contagious disease great care should be taken not to introduce it into the flock. When it is discovered in a flock it should at once be reported to the State Department of Agriculture and the flock quarantined. The affected animals should be separated from the uninfected and all should be dipped. The place where the sheep have been kept and all manure, litter, etc., should either be disinfected or, when practical, burned. Sheep introduced into a flock which is free of the disease should first be dipped if there is the most remote reason for suspecting that they came from an infected district.

Black Head, Head Scab, Black Nose

This form of scabies is much less frequent than the one just previously described. It is caused by a different mite, the technical name of which is *Sarcoptes scabei* var. *ovis*. This is a smaller parasite than the Psoroptic mite and burrows beneath the skin. A compound microscope is necessary for its definite recognition.

Symptoms. An itchy eruption occurs which confines itself pretty largely to the hairy parts of the face. It is usually found first on the lips, nostrils, eyelids and ears. Later on it affects the forehead, the intermaxillary space and cheeks. If it continues for any length of time it may spread to other parts of the body especially to the ventral portions of the abdomen and the pastern region. The affected sheep rubs its face on its fore legs and scratches it vigorously with its hind legs, often producing abrasions.

Treatment. Taken in time this disease is easily cured. Wash the affected portions carefully with soap and water. Loosen up all crusts. Then apply the parasecticide. This may be any of

a number of agents as lysol, 2–3 per cent., oil of tar, sulphur iodide and oil, 1–8 per cent., creolin, 2–3 per cent. Care should be taken to remove the flock to a non-infested pasture or sheepfold or the thorough disinfection of that in which they have been kept. All rubbing places, stones, fences, trees, etc., should be thoroughly disinfected.

Prevention. Great care should be taken to avoid adding animals affected with this disease to the flock. A careful examination of the head will usually reveal the individual which is affected.

LICE (PHTHIRASIS)

Lice are not found as commonly on sheep as on some of the other domestic animals, especially cattle. They are not a serious menace to the sheep industry of this State.

There are two main classes of lice, the biting and the sucking lice. The biting lice which belong to the genus technically known as the *Trichodectes* are usually smaller and lighter in color than the sucking lice. The mouth parts are arranged in form of mandibles which are short and usually serrated. They bite the skin of their host. The head is short and broad. The sucking lice, technically known as the genus *Haematopinus*, have their mouth parts arranged for sucking. They have a long narrow head, a protractile proboscis. Within is a piercing stylet for perforating the skin of the host and drawing blood.

The principal species of lice found on sheep are as follows: The sheep foot louse (*Haematopinus pedalis*) a sucking louse which is found on the lower part of the legs. The biting louse (*Trichodectes ovis* — (*sphaerocephalus*)) which may be found on nearly any part of the body.

The lice are easily recognized at the time of shearing and are most easily destroyed at this time. Sheep lice are quite small, being about 1/25 to 1/20 of an inch in length. The biting louse has a reddish head and pale transverse lines across the abdomen. The general color is a grayish white.

Symptoms. Infestation with these parasites leads to rubbing and itching. The wool gets matted. The lice are often in groups close to the roots of the wool and the adjacent skin shows reddish spots. The skin is also roughened with scales and debris.

Treatment. This consists in the destruction of the lice and their eggs known as *nits.* Almost any coal tar product as creolin, kresco or lysol in 2 per cent. solution will destroy the parasites. Pyrethrum may be employed as a dusting powder. Sulphur ointment may also be used. It must be remembered that the agents used to destroy the lice will not usually destroy the eggs and a second or third application must be made to rid the animal of the young lice which hatch.

Prevention. Keep all surroundings of paddock, sheep pen, etc., clean. Wash with a good disinfectant and whitewash. Lice are usually associated with filth.

Sheep Gadfly — Grub in the Head

This is a very common parasite and causes sheep much annoyance. It is the larva of this parasite which inhabits the nasal chambers of the sheep and causes severe inflammation. The fly is a little larger than the common house fly, brownish or yellowish in color and hairy. The abdomen is variegated with brown and straw yellow and is found usually curved under toward the head. The wings are transparent and are marked at their base by three black points. It has no mouth and therefore cannot take nourishment.

The fly attacks sheep from June to October and in warm stables may continue to deposit its larva during the winter months. The larva is deposited at the margin of the nostrils and by means of hooks attaches itself and gradually works its way up into the nose. They may crawl up as far as the base of the horns in rams. They attach themselves to the mucous membranes of the nasal and maxillary sinuses and to the turbinated bones. They remain here about ten months. After having attained maturity they pass downward or outward into the nose and are expelled by sneezing. The larva bores its way 1 or 2 inches into the ground, passes to the pupa state and in from three to eight weeks emerges as an adult fly. It gradually grows and attains a length of ¾ to ⅞ of an inch. The larva when deposited is about 1/12 of an inch long, cylindrical, tapering at each end and is made up of eleven rings. It is smooth on one side and provided with short spines on the other and has two hooks at the mouth. It is at first white, then changes to a yellowish hue and finally becomes brown in color.

Symptoms. When attacked by these flies sheep become frantic. They run wildly, striking at their noses with their front feet, rubbing their noses in the ground or hiding them under other sheep. They shake their heads and snort. Sheep will often crawl under trees or in fence corners or caves to escape the attack of these insects. If available, they will find a dusty place where they raise a cloud of dust which keeps the flies away. If the larva has been deposited and works up into the sinuses, a discharge appears from the nostrils which is at first clear and later becomes nearly pure pus. Swelling of the throat and nostrils is seen in heavy infestations. When the sheep moves about it may lift its feet high as if walking in water or it may move unsteadily or even fall. In a majority of cases when the animal is affected with only a few larvae the disease is not fatal and the only symptom noted is a nasal discharge.

In some instances this infestation has been confused with Gid or infestation with the larva of *Multiceps multiceps* (*Coenurus cerebralis*) which is found in the brain. In this latter disease, however, there is no nasal discharge, no rubbing of the nose and no larvae or grubs are discharged from the nasal cavities.

Treatment. A variety of medicinal agents have been used to dislodge the grubs, these being injected into the nasal cavities. Tobacco water, oil of turpentine, olive oil, etc. are among those most commonly used. They are of but little value except in the very early stages or when the larva has not become firmly attached. The best agent we have used for the purpose of dislodging the grubs is gasoline, which is injected by a syringe with a long nozzle or may simply be poured into the nostril with the sheep lying on its side. This treatment may be repeated at intervals or until all the grubs are expelled.

Surgical measures have been employed in the treatment of this disease. They consist simply in trephining into the frontal sinuses and injecting some one of the agents mentioned above.

Prevention. Keep sheep away from infested or shrubby pastures. If this is impossible smear the noses of the sheep with some agent that will keep the flies away. Tar or tar and oil or better, a mixture of tar, naphthalin and lard should be smeared on the nose every few days during the summer months and until October.

Internal Parasites

Stomach Worm Disease

This disease is the most serious that affects sheep in this State. The losses due to the ravages of this parasite are little appreciated. Many flocks have been nearly exterminated due to infestation by this worm. As stated before, we have made autopsies on a number of sheep from several flocks in the regular post-mortem work of the college and in nearly every case we have found the cause of death to have been this parasite. We wish to strongly emphasize the importance of this disease as an economic feature in the sheep raising of the Empire State. This worm seems to have gained a foothold here and the veterinarian must do all in his power to prevent its further spread and bring to this work the latest information obtainable. To illustrate what other countries are doing to combat this pest we will mention the Union of South Africa. This locality, which is a part of the British Empire, is one of the largest sheep producing districts in the world. In 1911, under the stimulus of Sir Arnold Theifer, Director of Veterinary Research, Dr. Veglia began an extensive series of researches concerning this parasite. In the first place the anatomy, morphology and cycle of development of this parasite was studied. The results of these studies have just been published. New experiments are being conducted to attempt to discover the best medicinal treatment. This work has extended over a period of six years and has cost considerable money but it is well invested if this parasite can be controlled as the result of these studies.

Cause. The worm which causes this disease is technically known as *Haemonchus contortus.* It is found in the fourth stomach of the sheep. The male is from ½ to ¾ of an inch in length, the female ¾ to 1¼ of an inch. The diameter is from 1/32 to 1/16 of an inch. The color is red or white according as to whether the intestine is filled with blood, sucked from its host. It takes the name contortus from the fact that the ovarian tubes are wound around the intestine in the female. The ovarian tubes are white while the intestine is dark in color giving a rather distinctive appearance. The mouth is provided with a lancet shaped apparatus which pierces the mucous membrane of the stomach, permitting the parasite to suck blood.

The eggs of this parasite are very small, oval and measure on the average 70.9 μ by 45.9 μ. In fresh feces they have a transparent shell about 1 μ or less in thickness. A freshly laid egg is usually found in the four cell stage. They hatch in from a day or two to several weeks, depending on the conditions of temperature and moisture. Drying kills the larva rather quickly. In a laboratory experiment carried out by Veglia, he found that mature larvae lived for five months in water, while in dry feces in the same room 50 per cent. of the larvae were dead in eight days. The newly hatched larva feeds on the fecal matter in which it finds itself. It passes through these stages of development and in the third stage is a mature larva. These are a little more than one-half millimeter in length. The mature larvae crawl on to the blades of grass under favorable conditions of moisture, light and temperature. The sheep take them in while grazing. The mature larvae during cold weather bore into the ground and are able to pass through the winter without heavy mortality.

The larva when taken in with the food enters the stomach, passes here through another moult and becomes an adult worm on the average in fifteen days.

Symptoms. The sheep gradually lose in flesh. A general anemia is the result of an infestation by this worm. The visible mucosae are pale, the wool looks unthrifty and general cachexia results. Toward the later stages of the disease convulsions and nervous symptoms are noted. The animal staggers, is unable to walk in a straight line and often opisthotonos is manifested. In the living animal the proper and only way to make a positive diagnosis is by the finding of the ova of these parasites in feces. This can easily be done by mixing the feces with some water to dilute them and then putting a drop on a glass slide over which place a cover glass and examine under the microscope.

If an animal is recently dead, a post-mortem should be done to determine positively the presence or absence of the worms. One thing should be borne in mind in connection with autopsies on sheep. It has been our experience that post-mortem decomposition takes place more rapidly in sheep than in the other domestic animals. Care must be taken not to confuse post-mortem changes with those that occurred before death. Furthermore, in the

autopsies done by us on sheep which we knew were affected by this parasite and which had undergone post-mortem decomposition, it was only with the greatest care that we were able to find the worms. It would have been a very easy mistake to have missed them altogether. It is far better if this disease is suspected, to kill one of the animals that shows the most pronounced symptoms and autopsy immediately.

The more common changes are anemia of the subcutaneous tissues, mucous membranes and the internal organs. The blood is lighter in color than normal. Emphysema of the subcutaneous connective tissue has been noted. The mucosa of the abomasum is spotted with hemorrhages (petechiae and ecchymoses). The contents are dark in color and contain blood. Masses of the worms are found here, some attached to the mucosa. If the animal has been killed or recently died the worms are very active, swimming around in the stomach contents rapidly. In the post-mortems done at the college this year we have uniformly found fatty degeneration of the liver and kidneys.

Treatment. Nearly every parasiticide in the pharmacopoea has been used in the treatment of this disease. The ones to be mentioned have given the best results.

In the Ambulatory Clinic, the copper sulphate treatment recommended by the South African authorities and extensively used by them has given the best results. One pound (avoirdupois) of copper sulphate is dissolved in 9½ gallons of water. The copper sulphate had better be dissolved in 2 or 3 quarts of hot water first and the remainder added cold. The doses recommended are as follows:

Lambs three months old, ¾ ounce.
Lambs six months old, 1½ ounces.
Sheep twelve months old, 2½ ounces.
Sheep eighteen months old, 3 ounces.
Sheep two years old, 3½ ounces.

Creosote is often used. A 1 per cent. solution in water is prepared by shaking and 2 to 4 ounces are given to lambs, 3–5 ounces to sheep. Care must be used in using nothing but pure beechwood creosote. Oil of turpentine and gasoline have also been

widely used. Dr. Law recommends arsenious acid 1 ounce, iron sulphate 5 ounces, powdered nux vomica 2 ounces, powdered areca nut 2 ounces, common salt 4 ounces. Divide into thirty powders and give one daily.

Prevention. This consists in keeping sheep and especially lambs from infested pastures. Many authors recommend rotation of pastures in one form or another. The following is taken from Ransom's work:

"It is evident from the foregoing statements that in the northern part of the United States, under usual climatic conditions, infested and non-infested sheep may be placed together in clean fields the last of October or first of November and kept there until March or even later, according to the weather, with little or no danger of the non-infested sheep becoming infested. If moved then to another clean field they may remain there nearly the entire month of April before there is danger of infection. From the first of May on through the summer the pasture becomes infectious much more quickly after infested sheep are placed upon them, and during May it would be necessary to move the sheep at the end of every two weeks, in June at the end of every ten days, and in July and August at the end of each week, in order to prevent the non-infested sheep from becoming infected from the worms present in the rest of the flock. After the first of September the period may again be lengthened. This method of preventing infection in lambs would require a considerable number of small pastures or subdivisions of large pastures, and in many instances could not be profitably employed, but in cases where it could be used it would undoubtedly prove very effective. By the time the next lamb crop appeared the pastures used the year before would have remained vacant long enough for the infection to have disappeared, and would consequently again be ready for use. By continuing this rotation from year to year, not only would each crop of lambs be protected from infection, but as reinfection of the infested ewe flock is prevented at the same time, the parasite would in a few years be entirely eradicated from the flock and pastures.

"If such frequent rotation is not possible or practicable, a smaller number of pastures may be utilized, after the ewe flock has been treated with vermifuges. The treatment may be given either before or after the birth of the lambs. If before, the ewes should be treated before pregnancy is too far advanced, in order to avoid possible bad results from the handling necessary in treatment. Probably the best time for treatment is late in the fall or early in the winter. The treated sheep should be placed immediately on clean pasture in order to avoid reinfection. The object of treating the ewes is to get rid of the worms with which they are infested, and thus remove the source from which the pasture becomes contaminated. If it were possible by treatment to free the old sheep entirely from stomach worms, it is evident that the lambs would remain free from infection, provided, of course, that the flock were afterwards kept on clean pasture. Unfortunately, there is no vermifuge known which can always be depended upon to remove all of the worms, but it is possible to get rid of most of them and thus greatly reduce the amount of infection to which the lambs will be exposed."

Nodular Disease

This is an exceedingly common affection among sheep and although not usually fatal nevertheless if the number of parasites becomes very numerous, the sheep loses in condition and finally may die. Further, this condition is sometimes confused with tuberculosis which is a very rare disease among sheep.

Cause. The worm which causes this condition is technically known as *Oesophagostoma columbianum.* The larval form of the parasite is found in nodules in the wall of the intestine. It is most frequent in the wall of the caecum but occurs in the other portions of the intestinal tract as well as in the liver and other viscera. The nodules are at first very small but gradually increase in size until they reach one-half an inch or more in diameter. They are irregularly spherical in shape. When the nodule is cut open it is found to be filled with a greenish, cheesey material upon which the larva feeds. When the larva has reached a certain stage of development it bores through the mucosa of the intestine and passes to the lumen where it develops to maturity. It is

not uncommon to find nodules having undergone calcareous degeneration.

The adult worm is small and whitish in color. The male is about one-half inch in length and the female a little longer. The body is narrowed at both ends. The ova are elliptical and vary in size from 65 by 40 μ to 75 by 45 μ in diameter. A positive diagnosis of this infestation is best made on post-mortem examination by the presence of the nodules which are easily observed projecting from the serous surface of the intestines.

Symptoms. The symptoms of this disease are not definite and might be assigned to other causes. They consist of emaciation and diarrhea and general debility. The sheep do not develop as they should, the general condition becomes poor and post-mortem examination shows large numbers of the characteristic nodules. From the uniformity with which nodules are found on the intestines of sheep in good condition, it is reasoned that a mild infestation does the host very little if any injury. When large numbers are present, however, they lead to digestive derangement.

Treatment. Medicinal agents have very little if any effect on the larval stage of this parasite. It is this stage (larval) that does the greatest harm to the host and for this reason drugs are of very little value in this disease. However, the adult forms of this worm can be expelled by using the ordinary vermifuges, such as turpentine, creolin, santonin, tartar, etc.

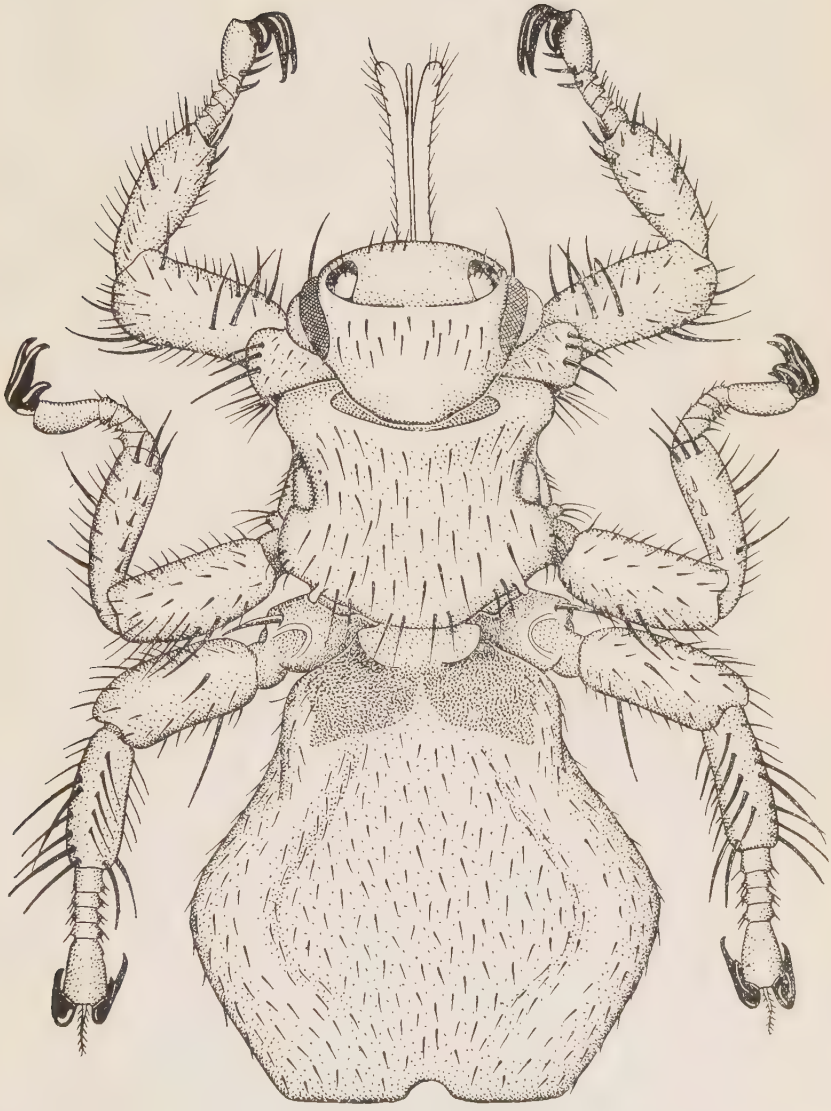
Prevention. This can be done only by change of pasture and the thorough cleansing of all materials contaminated by the feces. Ransom recommends that lambs be protected from infestation by these parasites by the so-called "bare lot" method. In this method the lambs and ewes are kept in enclosures free from vegetation and fed and watered from racks and troughs arranged so that they cannot be contaminated by the feces of the infested ewes.

There are a number of other parasites which infest sheep but they are of less importance and less commonly met with than the foregoing. In European countries and in certain parts of the United States the ravages produced by the liver fluke, *Distoma hepatica*, causing liver rot, are of great economic importance. Also in this same connection should be mentioned *Multiceps multiceps*, the larval stage of which produces gid and *Taenia*

echinococcus, which causes the so-called "hydatid disease." These parasites are rare in this State although a few outbreaks of gid have been reported. Another parasite which causes great loss in the west is the fringed tape worm, *Thysanosoma actinioides* (*T. fimbriata*). This parasite is found in the intestine and often works its way to the liver through the bile ducts. This parasite is sometimes introduced into this State by sheep shipped from infected districts. We have found it on post-mortem examination at the college in sheep coming from Wyoming. The veterinarian should, therefore, be on the watch for it in sheep coming from the western districts.

Lung worm disease or verminous bronchitis is not common among sheep of this State. It is, however, occasionally found. The parasite causing this disease is *Dictyocaulus filaria* (*Strongylus filaria*). Herms and Freeborn of California have studied this parasite extensively, especially as regards treatment with chloroform. Their report can be found in Circular No. 148, University of California, Berkeley.

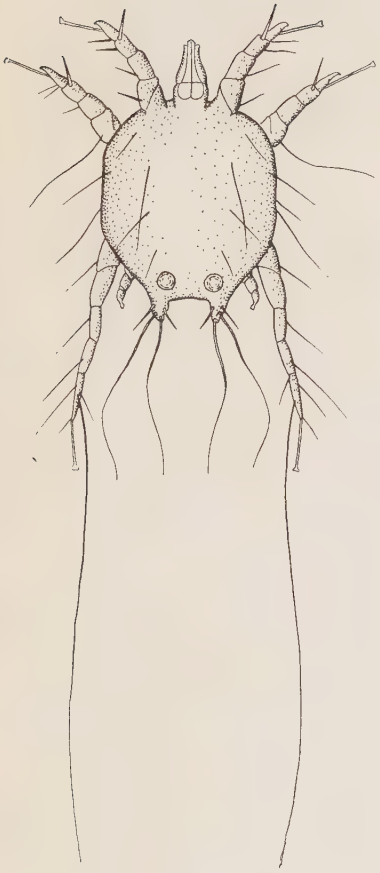
PLATE I.



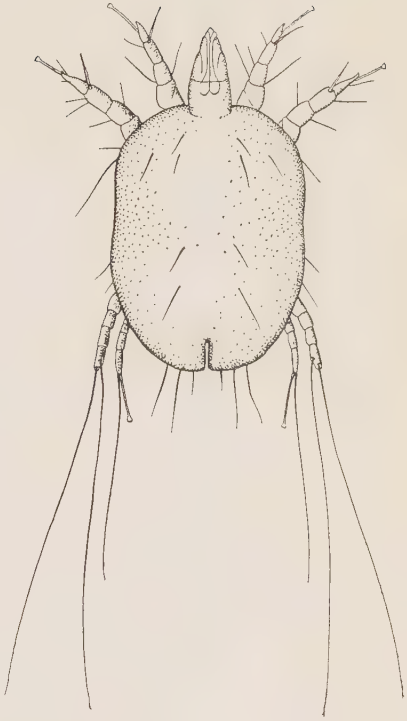
Melophagus Ovinus (Sheep Tick) — Original

PLATE II.

Male



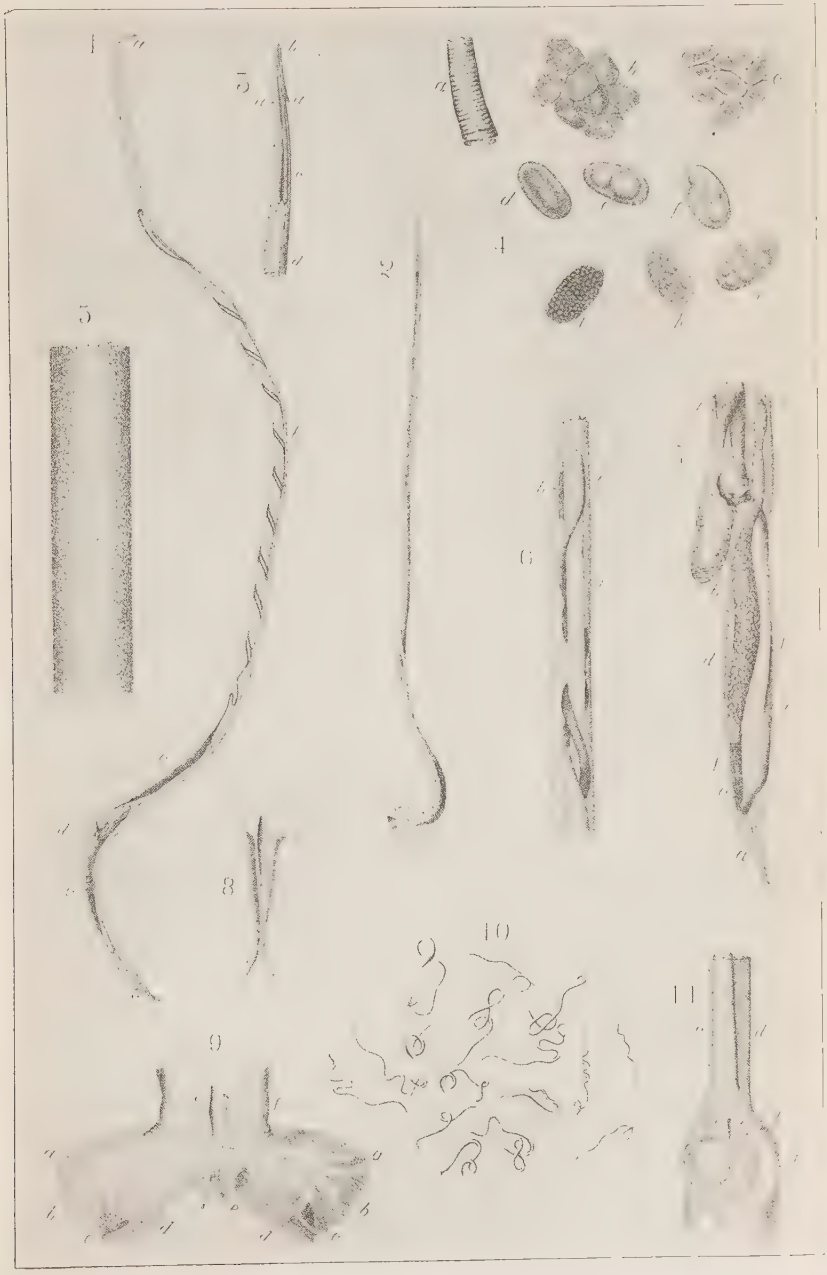
Female



Psoroptes Communis Ovaris. The Mite Causing Sheep Scab – Original

PLATE III.

1. Adult female
2. Adult male
3. Head
4. Eggs in different stages of development
5. Skin
7. Caudal end of female
8. Spicula
9. Male, caudal pouch
10. Group of adult males and females, natural size
11. Caudal end of male



HAEMONCHUS CONTORTUS (The twisted stomach worm)

PLATE IV.

Photograph of Intestines Affected with Nodular Disease — One Nodule in Center Incised — Original

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